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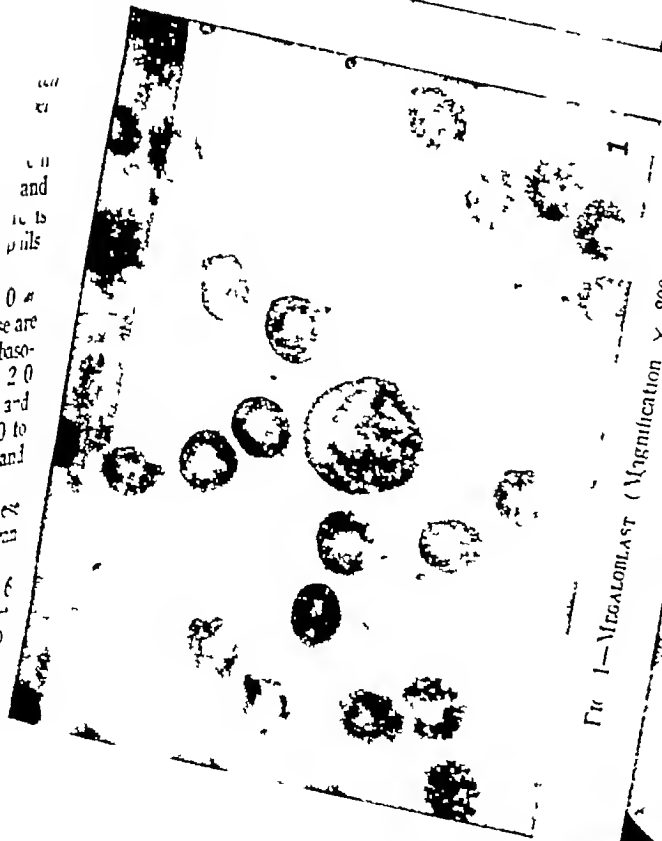


FIG 1—MEGALOBLAST (Magnification $\times 800$)

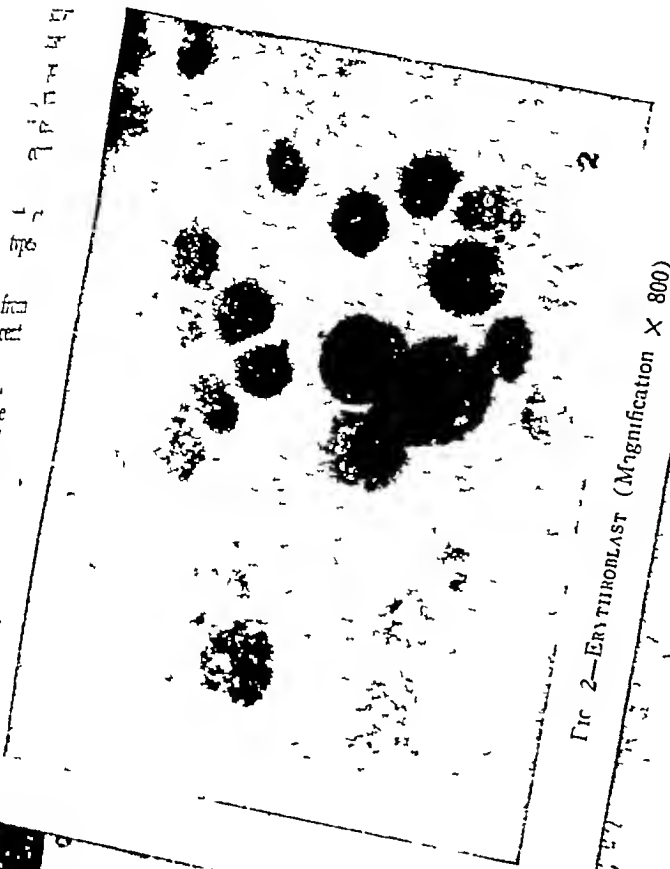


FIG 2—ERYTHROBLAST (Magnification $\times 800$)

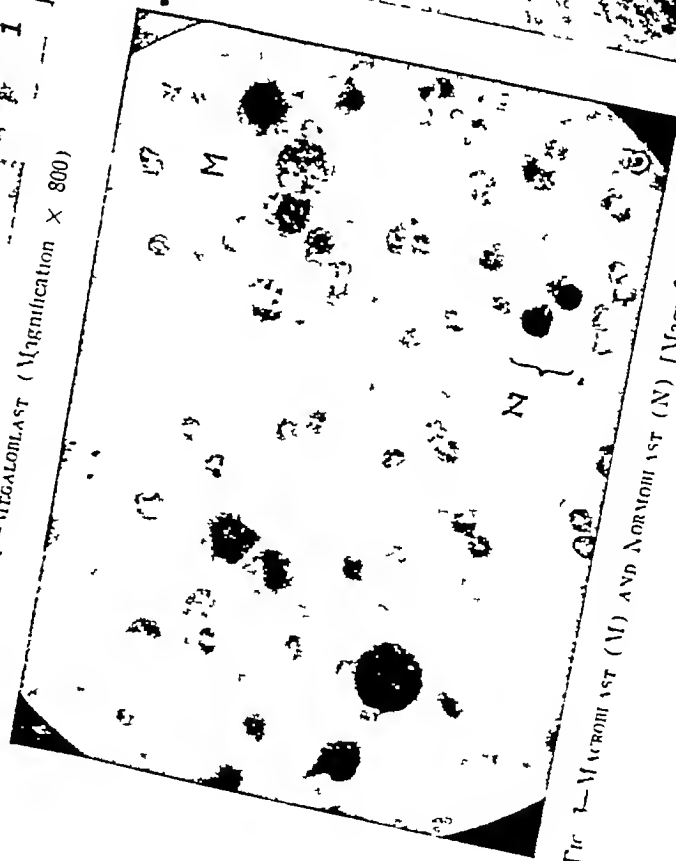


FIG 3—MACROMAST (M) AND NORMOMAST (N) [Magnification $\times 800$]

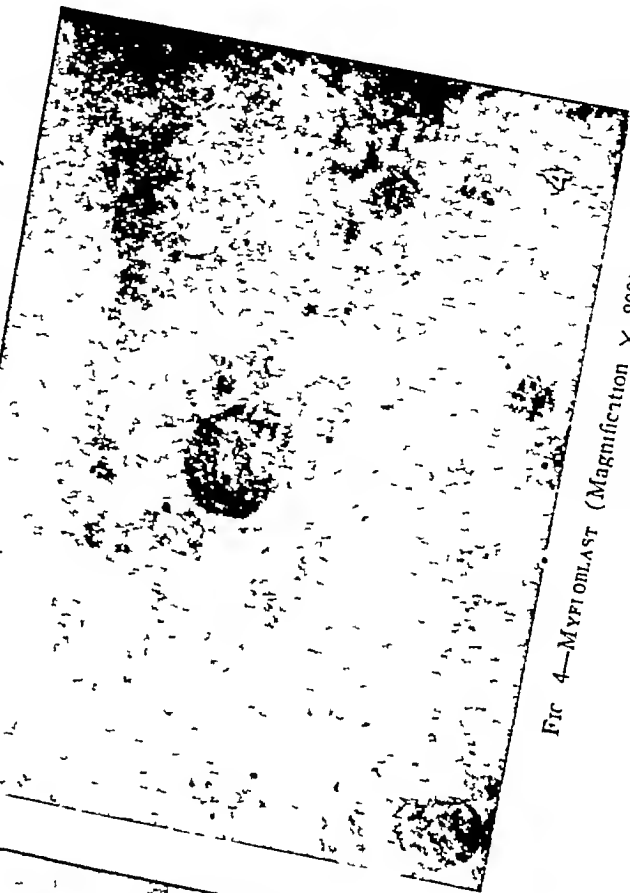


FIG 4—MYELOBLAST (Magnification $\times 800$)

rest of the fluid was put into an oxalated tube, potassium and ammonium oxalates (0.08 cc of potassium and 0.12 cc of ammonium oxalates of 1 per cent solutions for 1 cc of blood) being used to prevent coagulation. The tube was dried in hot air sterilizer. The smears were stained with Læshman's stain.

The oxalated specimen was examined for —

(a) *Enumeration of total nucleated cells, following the method detailed by Beck (1938)*

(b) *Estimation of hæmoglobin using the Hellige normal hæmometer as described by Beck (1938)* Hellige 100 per cent 13.75 gm per 100 cc

(c) *Enumeration of red cells using the new improved bright line Neubauer counting chamber and the technique of Beck (1938)*

(d) *Estimation of reticulocyte percentage by the method of Osgood and Wilhelm (1940) as modified by Napier (1940)*

(e) *Estimation of packed cell volume by Beck's technique (1938) using hæmometer tube with an electric centrifuge of 25000-30000 revolutions per minute. The result is expressed in percentages.*

Accurate differential count of the different nucleated cells was made by counting five hundred or more nucleated cells from different parts of well-stained smears and several smears were used to show for an uneven distribution of various cell types. Identification of different cells was made by reference to Napier's tables (1938). Size of the primitive differentiated cells was measured with the micrometer, the average diameters are given in the table. Figs 1-8 are reproduced from photomicrographs of the red and white blood cells in marrow smears.

OBSERVATIONS

The results statistically computed are given in Tables IA and IB. The cell types are shown in Figs 1-7.

1 *Hæmoglobin in gm per 100 cc blood* There is a deficiency of 0.6 gm in the hæmoglobin content of the sinusoidal blood. It varied from 12.51 to 15.9 with the mean 14.35 and S.D. 0.9575.

2 *Red cells in millions per cmm blood* The sinusoidal blood is deficient by 0.468 million per cmm. It ranged from 4.05 to 5.98, with a mean of 4.61 and S.D. 0.4722.

3 *Reticulocytes percentage of red cells* High in the sinusoidal blood but the increase is not appreciable. It ranged from 0.2 to 2.0 with the mean 0.79 and S.D. 0.33.

4 *Total nucleated cells per cmm* This varied from 17600-112780 with a mean of 47700 per cmm and S.D. 19900. The count varied a great deal in different punctures of the same person and in the successive portions of the same specimen. The marrow cells readily adhere to one another and to the sides of the syringe and the test tube if the blood is not thoroughly mixed with the coagulant. *This estimation should therefore be made immediately.*

5 *Volume of packed cells* This varied from 42 to 59 per cent with the mean of 48.46 per cent and S.D. 5.28. The sinusoidal blood was richer in this than the peripheral.

6 *Differential nucleated cell count* These results are given in Table IB and the cell types shown in Figs 1-7. The terminology of the cells is based on that of Whitby and Britton (1937). About 10 per cent of the cells are difficult to be classified along with well differentiated cells. The Fudge and smear cells are not taken into consideration in differential count when establishment a myelogramme.

A *Red cells* These form 19.34 per cent of the total nucleated cells and include the following types of cells. The counts are given in Table IB.

1 *Megaloblast (Fig 1)* Range is from 0 to 3 per cent with a mean of 0.61 per cent and S.D. 0.6145.

2 *Erythroblast (Fig 2)* Some workers have classified these cells as early and late erythroblast but here the latter are separated into the distinct group macroblast. They vary from 0 to 5 per cent with the mean of 1.63 per cent and S.D. 1.0040. Size 12-8 μ .

3 *Macroblast (Fig 3)* These vary from 1 to 11 per cent with the mean of 4.76 per cent and S.D. 2.4183. Size 7 to 14 μ .

4 *Normoblast (Fig 3)* Range from 4 to 20 per cent with the mean of 12.34 per cent and S.D. 2.2758. Size 10 to 12 μ .

B *White cells series* These formed on an average 80 per cent of the total nucleated cells and consisted of about 61.42 per cent of cells of granular series and 10.12 per cent of non-granular series.

The granular series consisted of

1 *Myeloblast (Fig 4)* Range from 0.0 to 5.0 per cent with a mean of 0.58 per cent and S.D. 0.9558. Size 14.5 to 19.5 μ .

2 *Premyelocyte (Fig 5)* Range from 0.0 to 2.0 per cent with a mean of 0.60 and S.D. 0.4204. Size 14.5 μ to 20.0 μ . There is no differentiation of these cells into neutrophils, eosinophils and basophils.

3 *Myelocyte* Size 14.5 μ to 19.0 μ . According to the colour of the granules these are classified as neutrophils, eosinophils and basophils. The neutrophils varied from 0.2 to 2.0 per cent with the mean of 0.88 per cent and S.D. 0.3180 and basophils varied from 0.0 to 0.5 per cent with the mean 0.02 per cent and S.D. 0.1939 (Table IB).

4 *Metamyelocytes (Fig 6)* These varied from 0.0 to 13.5 per cent with the mean 5.50 per cent and S.D. 3.6691.

5 *Non-segmented polymorphs (Figs 6 and 7)* These varied from 18 to 55 per cent with the mean 33.64 per cent and S.D. 8.5669.

6 *Segmented polymorphs (Fig 8)* These varied from 6 to 30 per cent with the mean 17.48 per cent and S.D. 5.0645.

The non-granular series consisted of

1 *Lymphocytes* Varied from 5 to 39 per cent with the mean 18.12 per cent and S.D. 9.1017.

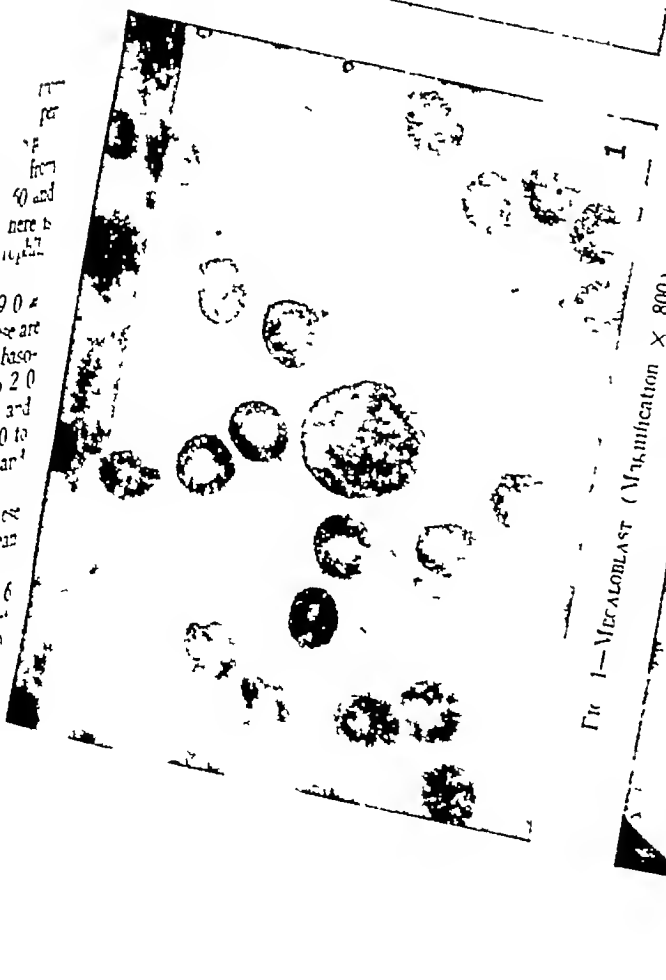


FIG 1—MEGALOBLAST (Magnification X 800)

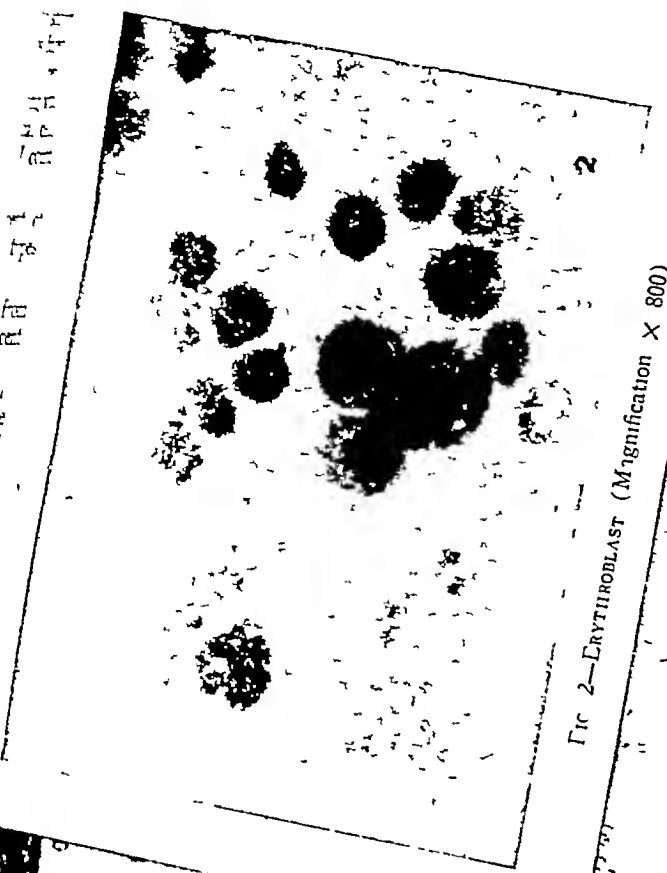


FIG 2—ERYTHROBLAST (Magnification X 800)

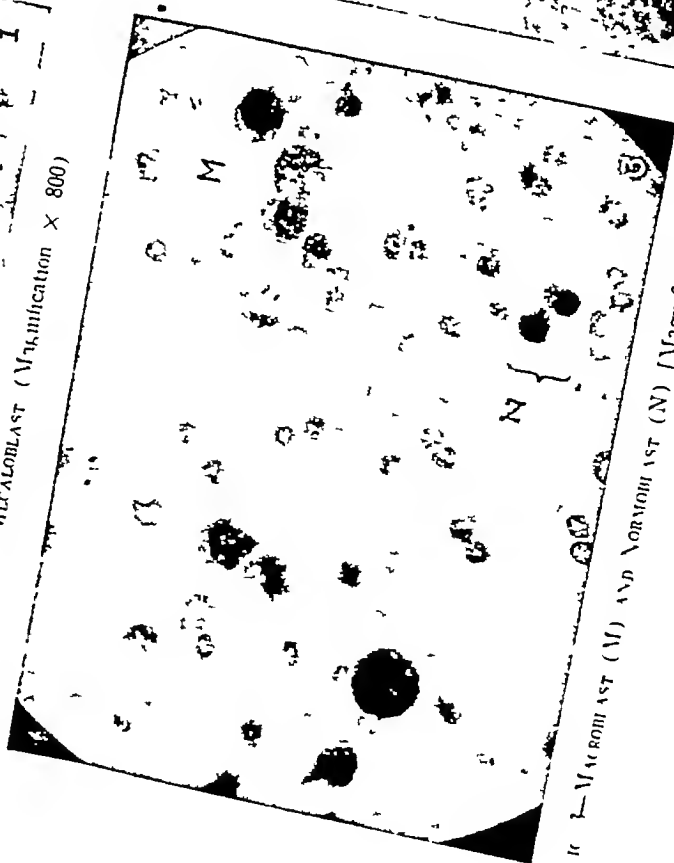


FIG 3—MACROMAST (M) AND NORMOBLAST (N) [Magnification X 800]

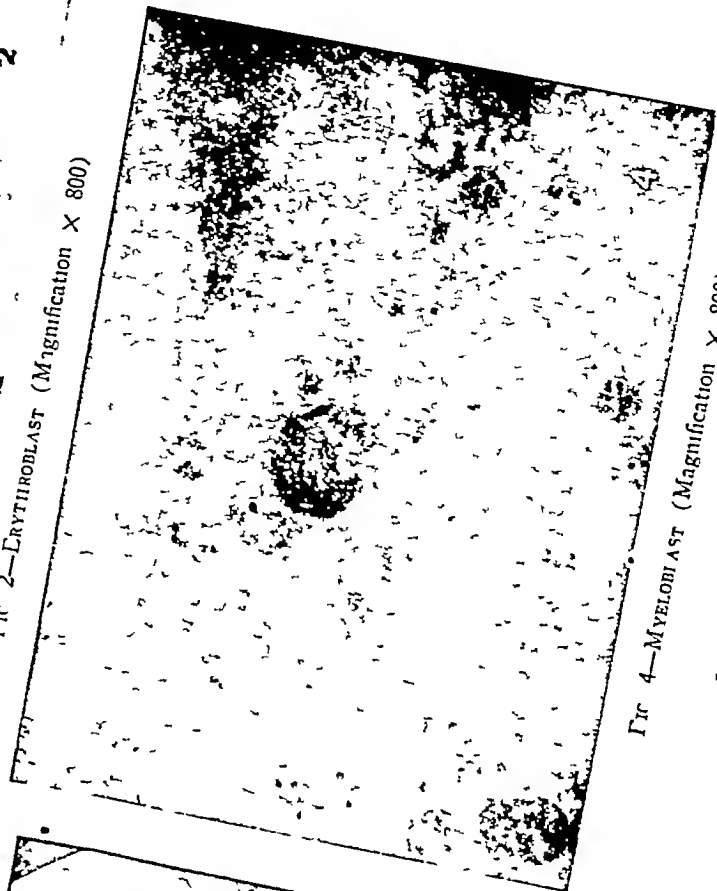


FIG 4—MYELOBLAST (Magnification X 800)



FIG 5—PROMYELOCYTE (Magnification $\times 800$)

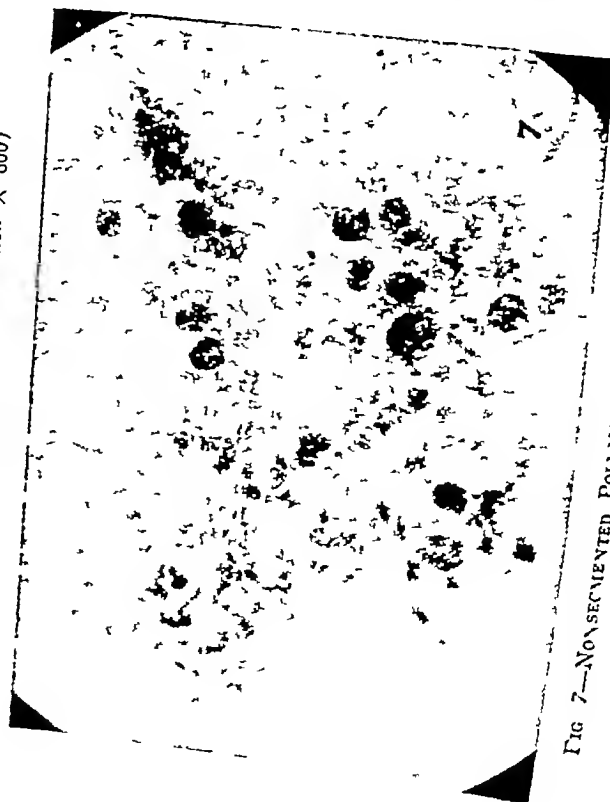


FIG 7—NONSEGMENTED POLYMORPHS (Magnification $\times 800$)



FIG 6—METAMYELOCYTES (Magnification $\times 800$)

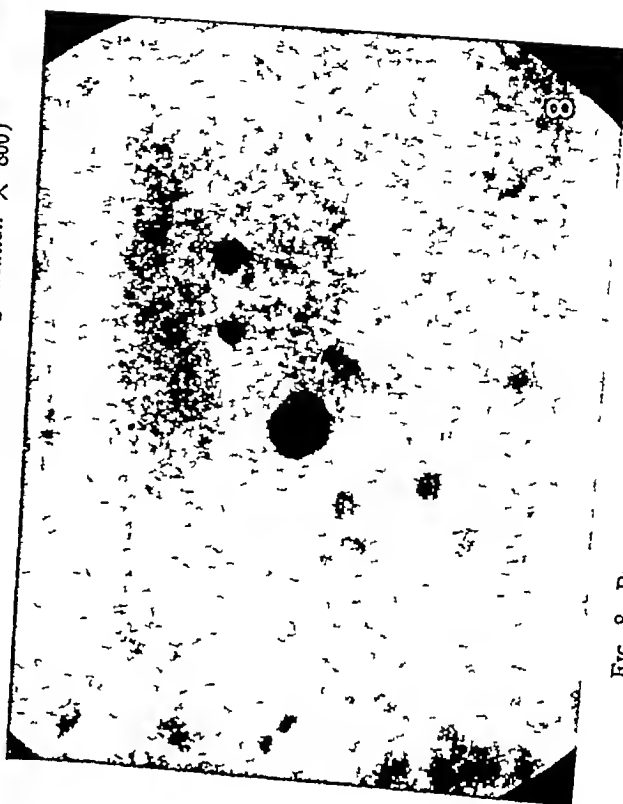


FIG 8—PLASMA CELL (Magnification $\times 800$)

2 Monocytes Typical monocytes varied from 0 to 5 per cent with the mean 0.88 per cent and S.D. 1.0514. No primitive monocytes are found.

3 Plasma cells (Fig. 8) Varied from 0 to 1 per cent with the mean 0.12 per cent and S.D. 0.6499. Size 14.

4 Megakaryocyte (Fig. 7) These were usually found at the edge of the smears. Size upto 53 μ . These are not included in the differential nucleated cell count.

C Myeloid/Nucleated Red Cell Ratio This varied from 1.62 to 8.3 per cent with the mean 3.92 per cent and S.D. 1.6620 (Table IA).

TABLE IA—SHOWING NORMAL BONE MARROW STANDARD (AUTHOR)

No	Items	Range	Mean	Standard deviation
1	Hæmoglobin in gm. per 100 c.c.			
	Peripheral blood	13.40—16.50	14.96	0.9048
	Sinusoidal blood	12.513—15.950	14.35	0.9575
2	Red cell per c.mm. in millions			
	Peripheral blood	4.40—6.00	5.08	0.3635
	Sinusoidal blood	4.05—5.98	4.612	0.4722
3	Reticulocytes per cent of red cells			
	Peripheral blood	0.2—1.8	0.589	0.2948
	Sinusoidal blood	0.2—2.0	0.796	0.3333
4	Volume of packed cells			
	Peripheral blood	38.56	44.26	3.60
	Sinusoidal blood	42.59	48.46	4.28
5	Total nucleated cells per c.mm.	17600—112780	47700	19900
6	Percentage of nucleated red cells in the total nucleated cells	6—30	19.34	3.0228
7	Percentage of leucocytes in the total nucleated cells	—	80.68	—
8	Percentage of granulocytes in the total nucleated cells	54—81	61.54	8.6396
9	Percentage of non-granulocytes in the total nucleated cells	5—45	19.12	1.6454
10	Myeloid/nucleated red cell ratio	1.62—8.3	3.92	1.6620

TABLE IB—SHOWING NORMAL BONE MARROW CYTOLOGY (AUTHOR)

Items	Range	Mean	Standard deviation
A Nucleated red cells			
Megaloblast	0.0—3.0	0.61	0.6145
Erythroblast	0.0—5.0	1.63	1.0040
Macroblast	1.0—11.0	4.76	2.4183
Normoblast	4.0—20.0	12.34	2.2758
B White cell series			
Myeloblast	0.0—5.0	0.58	0.9558
Premyelocyte	0.0—2.0	0.60	0.4204
I Granular Series			
Myelocyte			
Neutrophil	0.2—2.0	0.88	0.4633
Eosinophil	0.0—0.01	0.88	0.3180
Basophil	0.0—0.5	0.02	0.1939
Metamyelocytes	0.0—13.5	5.59	3.6691
Non segmented Polymorphs	18.0—55.0	33.64	8.5660
Segmented Polymorphs	6.0—30.0	17.48	5.0545
Eosinophil (Mature)	0.0—6.0	1.80	1.5472
Basophil (Mature)	—	0.50	—
II Non granular series			
Lymphocytes	5.0—39.0	18.12	9.1017
Monocytes	0.0—5.0	0.83	1.0514
Plasma cells	0.0—1.0	0.12	0.6499

TABLE 2—SHOWING THE NORMAL BONE MARROW CYTOLOGY ACCORDING TO DIFFERENT OBSERVERS

<i>Cells</i>	<i>Schilling & Benzlar (1915)</i>	<i>Arunkin (1929) Average</i>	<i>Holmes & Brown (1933) Average</i>	<i>Tempka and Brown (1932) Range</i>	
Megaloblast			5 2		
Erythroblast & Normoblast	37 45	12 7	6 9	13 3—16 6	
Myeloblast		1 7	2 4	4 6—7 0	
Pre-myelocyte		1 9		3 7—6 8	
Myelocyte					
Neutrophil	17 39	6 55	7 0	12 7—13 3	
Eosinophil		0 65		1 5—2 7	
Basophil				0 0—0 3	
Neutrophil					
Young	18 98	2 4	6 7	14 3—16 2	
Band	0 11		14 0	17 0—22 5	
Segmented	4 85	48 0	17 4	16 2—20 3	
Eosinophil	2 91	2 31	1 0	1 5—7 2	
Basophil		0 35	0 3	0 2—0 7	
Lymphocyte		11 9	24 9	2 6—3 3	
Monocyte	18 96	5 7	9 0	0 5—0 8	
Plasma cell		0 6		0 3—1 6	
Megakaryocyte					
Disintegrating cells		3 35		2 2—4 0	
Reticulocyte percentage of red cells		0 8—1 4		0 8	
Myeloid/Erythroblastic ratio		7 7 to 4 17 1	4 86 1 0	5 83 1 to 5 39 1	
<i>Cells</i>	<i>Young & Osgood (1935) Range</i>	<i>Segerdahl (1935)</i>	<i>Vogt Erf & Rosenthal (1937)</i>	<i>Napier & Sen Gupta (1938)</i>	<i>Author's series Average</i>
Megaloblast	0 0—4 2		7 1	0 7	0 61
Erythroblast & Normoblast	5 4—20 0	12 88	22 6	25 0	18 73
Myeloblast	0 0—1 2	1 32	1 6	1 2	0 58
Pre-myelocyte	0 0—7 8	1 35	0 1	0 7	0 60
Myelocyte					
Neutrophil	0 0—2 6	15 00	21 5	4 4	0 88
Eosinophil	0 0—0 4	1 37	2 1	1 3	0 88
Basophil					0 02
Neutrophil					
Young	1 8—9 8	15 69		9 7	5 60
Band	15 8—33 0	10 48	30 2	25 3	33 64
Segmented	7 4—25 2	20 86	34 0	16 8	17 48
Eosinophil	0 0—4 6	1 44	0 77	4 7	1 80
Basophil	0 0—0 8	0 14	1 33	0 2	0 50
Lymphocyte	4 8—16 0	16 71	8 6	6 25	18 12
Monocyte	0 0—4 2	2 27		3 0	1 05
Plasma cell	0 0—1 0	0 39		0 6	0 65
Megakaryocyte	0 0—0 2	0 029	0 2		
Disintegrating cells	12 8—31 8	0 031			
Reticulocyte percentage of red cells	1 2—5 4		0 25	0 75	0 80
Myeloid/Erythroblastic ratio	8 29 1 to 2 00 1	4 1 1		2 97 1	8 3 1 to 1 62 1

DISCUSSION

The largest single series of normals is that of Segerdahl (1935) who performed sternal puncture on 52 young men, 40 young women, nine old-men, and nine old women. There is no great difference between any of these groups. Unfortunately Segerdahl's classification of the cells differs considerably from that used here particularly as regards myelocytes and metamyelocytes and it is therefore not possible to make use of her very complete statistical analysis. There does not appear, however, to be any gross discrepancy between her figures and the normal range suggested in the present series.

The number of leucocytes and nucleated red cells ed together in the marrow fluid is a rough indi-

cation of the cellularity of the marrow. The count depends also, however on the amount of blood mixed with the fluid, the first drop of fluid has a very good marrow content, whereas if several ccm are withdrawn, the later portions may contain hardly any marrow. *It is therefore important to withdraw a small and constant volume of the fluid.* If much blood is withdrawn, the total cell count is low and the percentage of segmented polymorphs and lymphocytes is unduly high, the percentage of other cells being proportionately decreased. Such findings suggest the need to repeat the puncture. In this series the total cell count ranged between 17600 and 112780 per cmm with a mean of 47700 and S D 19900. Segerdahl's (1935) range was much larger, from 10600 to 238200.

per cmm with a mean of 75000, and S D 38400 per cmm and Zanaty's (1937) range was very similar Napier's (1938) range was 25500 to 80000 in first series of ten perfectly normal individuals while in second series of filaria cases it was from 47000 to 175000 which is slightly higher. A total cell count below 20000 cmm strongly suggests a hypoplastic marrow, the one which is above indicates hyperplasia.

Segerdahl found no significant difference in the marrow cell count according to age and sex. By histological studies however Custer and Ahlfedt (1932) found the proportion of red marrow in the sternum to decline fairly evenly from 100 per cent at one year to 44 per cent at seventy years of age. In this series it was noticed that there is diminution in the cellular contents of the marrow as age advances but the number of cases examined in fourth and fifth decade was not sufficient to finally warrant any conclusion. In analysing sternal puncture counts most workers have taken the proportion of granulocytes to erythroblastic cells as an important figure, as it is convenient to express. This is often termed the myeloid/erythroid ratio. This myeloid/erythroid or erythroblastic ratio indicates whether the granulocytes or red cells are most effected by a pathological process and in what relative degree. In this series the myeloid/erythroblastic ratio lay between 1.62 and 8.31 with a mean of 3.921. Young and Osgood (1935) range was 2.0 to 8.31.

Pontoni (1936) has suggested that a more accurate indication of the relation between granulopoietic and erythropoietic activity might be given by the ratio between the percentage of erythroblastic cells and that of the immature cells of the granulocyte series from myeloblast to young form neutrophil inclusive. Thus he has called the leuko-erythrogenetic ratio. The mean normal from present series is 2.1 and that of Scott's (1939) was 1.97.

Two other methods of analysis are sometimes used in the estimation of the myeloid and erythroblast maturity dispersions. These are expressed by stating the percentage proportions of myeloblasts, promyelocytes, neutrophil myelocytes, and metamyelocytes, taking the sum of the percentage of these cells in the total count as 100. In the erythroblast series the percentage of the total nucleated red cells represented separately by megakaryoblast, erythroblast, macroblast and normoblast express the maturity dispersion. These figures provide a means of assessing the degree of shift in the maturity of the granulopoietic and erythropoietic cells. The mean normal values calculated from present series are myeloblast 6.8 per cent, promyelocyte 7.1 per cent, myelocyte 20.2 per cent, metamyelocyte 65.9 per cent, megakaryoblast 3.1 per cent, erythroblast 8.4 per cent and macroblast and normoblast together 88.5 per cent respectively. These figures agree with those obtained from sternal puncture by Pontoni (1936) and Scott (1939), and with those

obtained from sternal trephine material by Escudero and Varela (1932).

The leucoblastic cells represent a greater proportion of the marrow cells than erythroblastic cells, on an average about 4 times (Table I) though peripherally the red cells are on an average 700 times more than the white cells which can be explained by the longevity of red cells and a short life of myeloid series of cells.

The normal bone cytology according to different observers is given in Table II. This shows that the author's results are in general agreement with those of the earlier observers and particularly Segerdahl, who used a mixed series of 110 normal individuals. The myeloid erythroblastic ratio also does not differ very much from that established by her.

SUMMARY

An investigation is made of the bone marrow from sternal puncture in a mixed series of fifty healthy individuals including labourers, middle class men and students whose ages varied from 10 to 50 years. The results are statistically analysed.

The normal bone standard (Table IA) and normal bone marrow cytology (Table IB) have been worked out. A critical comparison has been made of the author's findings with those of previous workers (Table II). The results while differing in minor points show a general agreement with those of the earlier workers.

REFERENCES

- ARIKIN, M. S.—*Folia hemat.*, 38 235, 1929.
BECK, R. C.—Laboratory Manual of Haematologic Technique 1930. W. B. Saunders Company.
CUSTER, R. P.—*Am. J. M. Sc.* 617, 1933.
DONOVAN, C.—*Trans. Bombay Med. Congr.*, 159, 1909.
ESCUDEO, P., AND VARELA, M. E.—*Haematologica*, 3 65, 1932.
HOLMES, W. F., AND BROWN, G. O.—*Proc. Soc. exp. Biol.*, 30 1306, 1933.
HYNES, M.—*J. Path. & Bact.*, 19 231, 1939.
NAPIER, L. E., AND SEN GUPTA—*Indian M. Gaz.*, 73 1, 1938.
NAPIER, L. E.—*Indian J. M. Research*, 28, 1940.
PEABODY, F. W.—*Am. J. Path.*, 3 179, 1927.
PONTONI, L.—*Haematologica*, 17 833, 1936.
READY, D. G.—*Indian M. Gaz.*, 74 664, 1939.
SCHILLING, V., AND BENZLAR, J. (1915), cited by SCHILLING, V.—*Deutsch. med. Wchnschr.*, 51, 1925.
SCOTT, R. B.—*Quart. J. Med.*, 8 127, 1939.
SEGERDAHL, E.—*Acta Med. Scand. Suppl.*, 64 1, 1935.
SEYFARTH, C.—*Deutsch. med. Wchnschr.*, 49 180, 1923.
STRUMI, M.—*Am. J. M. Sci.*, 177 676, 1929.
VOGEL, E., ERF, L. A., AND ROSENTHAL, N.—*Am. J. Clin. Path.*, 7 436, 1937.
WEIL, P. E., AND PERLES, S.—*La. Porcion Sternale Paris*, 1938.
WHITBY, L. E. H., AND BRITTON, C. J. C.—*Disorder of the Blood* 2nd Edn., 1937. London.
WILKINSON, J. F., AND ISRAEL, M. C. G.—*Quart. J. Med.*, 1940.
YOUNG, R. H., AND OSGOOD, E. E.—*Arch. Int. Med.*, 55 186, 1935.
ZANATY, A. F.—*Lancet*, 2 958, 1937.

INTESTINAL HELMINTHIASIS

D N DEBSARMA, M B,
Koilamari Tea Estate Hospital, Lakhimpur, Assam

During my practice for a number of years at different places of Assam I have come across a good number of cases of intestinal helminthiasis which is one of the common diseases in this country, and most of my patients were children, and their ages roughly ranged from 1 to 10 years. Majority of these little patients belonged to the poorer classes of people such as the tea-garden labourers. It goes without saying that the laws of sanitation and health are still in infancy amongst these poor people, their habits with regard to repast as well as to the disposal of night-soil are generally filthy. High incidence of the disease among the children is obviously due to utter lack of sanitary laws.

Cases of round-worms, thread-worms, hook worms and tape-worm are usually found in this part of the country. These loathsome creatures give rise to various manifestations in different systems of the human body, and these manifestations are often misleading and dangerous. Perforation of the bowel by the round-worms and their migration to the gall-bladder, eustachian tube etc are not very rare events (Savill). In this short communication an attempt will be made to show the importance of the worms as a cause of morbidity and mortality in our country, specially among the children, the features which have been frequently observed to be associated with the worm infections will be pointed out. Most of these features were present in my cases. The points have been briefly dealt with below under several headings.

DIGESTIVE SYSTEM

Appetite—Intestinal worms cause a capricious and sometimes ravenous appetite, in spite of which the victims (usually children) become thin and sallow (Savill). Unnatural desire to eat objects such as earth, kerosene oil etc., is a well-known feature in ankylostomiasis. Voracity in children with 'pot-bellies' is usually a character of worm infections.

Anorexia—Severe anorexia may occur in some cases due to the toxæmia caused by some unknown toxins generated by the worms and may lead even to fatal terminations in certain cases. A notable instance in experience of the author (1943) was that of a little lass of about five who apparently died of an intractable anorexia after her protracted illness due to worms.

Dyspepsia—Various digestive disorders like dyspepsia are caused by these parasites. They also interfere with the absorption of food and thus exert a very sinister influence on nutrition specially in children. Indigestion has been found to be a very common disorder among the children infested with worms.

Diarrhœa—Periodic attacks of diarrhœa may be caused by some intestinal worms. Acute diarrhœa often caused by the worms may simulate even cholera and typhoid (Debsarma, 1943).

Dysenteric symptoms—Worms may sometimes produce also dysenteric symptoms. These are due to irritation and ulceration of the intestines caused by the parasites (Majumdar, 1938). These dysentery-like symptoms were seen to have disappeared after anthelmintic treatment in several cases and can be distinguished from true dysenteries by microscopic examinations of stools in doubtful cases.

Hæmorrhage per anum—Intestinal hæmorrhage in small quantities may take place in helminthic infestations. The ankylostomum duodenale is a frequent cause of melæna (Savill, *loc cit*). The worms may also give rise to profuse bleeding per anum when they complicate some other diseases like typhoid fever.

Colic—Intestinal colic due to spasm of the bowels and mechanical pressure caused by masses of round-worms has been found to be a common occurrence both in children and adults who harbour these parasites in enormous numbers.

Intestinal obstruction—Masses of round-worms knotted together may cause intestinal obstruction and is indeed a very serious condition. Some amount of obstruction in the bowels by the knots of worms is quite possible in some cases.

Hepatic disorders—Various hepatic derangements like cholangitis and fatty degeneration (Rogers & Megaw, 1935) may occur in long standing cases of worm infections. The intestinal toxins secreted by the worms are probably responsible for these abnormalities. Many authorities hold that the worms produce their effects on the human system chiefly through the action of these toxins. In some instances the parasites themselves may produce some disorders during their passage through the organ.

Salivation—This is quite well-known to the physician. It is perhaps a reflex hypersecretion of saliva due to some gastric irritation.

Vomiting—Often times it occurs after a vermifuge which disturbs the worms and kills few of them and sets disintegrating them which produces a toxic condition giving rise to vomiting (Rajaram, 1944). Probably the toxins in the blood act directly on the vomiting centre in the medulla in such cases. Intense vomiting due to worms may be mistaken for that of pregnancy toxæmia in certain cases. Wandering of the worms to the stomach may also cause nausea and vomiting.

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Broncho-pneumonia—The larvæ of the ascaris passing through the lungs may cause it (Majumdar, *loc cit*). Some other worms like the ankylostomes also pass through the lungs and may cause pulmonary disorders of varying degree. The worms probably play some part in the causation of chronic bronchitis too. Respiratory catarrh is a common feature associated with prevalence of ascaris infection in children.

Asthma—Helminthiasis is often associated with asthma (Majumdar, *loc cit*). Paroxysms of asthma are sometimes seen in children with worm infestations. The worms may secrete some protein which may be responsible for the condition.

Hæmoptysis—The ascaris may enter into the lungs and cause hæmoptysis (Majumdar, *loc cit*) But the worms should never be made solely responsible for the condition if all other possible causes of hæmoptysis are carefully ruled out

Suffocation—This is also not impossible if by chance the round-worms wander into the rima glottidis

CIRCULATORY SYSTEM

Anæmia—Anæmia leading to debility and dropsy in some cases is a prominent symptom in chronic helminthiasis especially in ankylostomiasis. The possibility of a helminthic infection must be borne in mind in obscure cases of anæmia (Savill, *loc cit*). Ingestion of blood by the worms, oozing of blood from the minute wound produced by them and hæmolysis are the probable causes of the anæmia. Some authorities regard this anæmia as being mainly due to a hæmolytic agent generated by the worms. Also malnutrition plays some part in the causation of the disease at least in some cases. The hæmoglobin percentage in such cases is usually very low, it may fall to 20 per cent or even less than that. During recent years I have come across a good number of anæmic patients, most of them responded well to iron and liver therapy after anthelmintic treatment.

Cardiac disorders—According to Rogers and Megaw (*loc cit*) in fatal cases of hook worm disease the heart is dilated and may be hypertrophied and syncope from cardiac dilatation is a most common complication. Attacks of paroxysmal nocturnal dyspnoea (cardiac asthma) are also not impossible in advanced cases of ankylostomiasis as both cardiac and renal derangements may occur in such cases.

Eosinophilia—This occurs in worm infestations and is associated with a corresponding diminution of the proportion of the polymorphonuclears (Rogers and Megaw, *loc cit*)

GENITO-URINARY SYSTEM

Disturbed micturition—Frequent micturition and enuresis are said to be associated with intestinal worms. These are probably the results of reflex nervous irritation. Enuresis in children is a troublesome condition often met with in practice.

Vulvitis and pruritus ani—Thread-worms often migrate to the vulva and anus and give rise to these conditions specially in small children. The infants get infected with these worms usually when they begin to eat a mixed diet.

Skin Manifestations

Itch-sore (ground itch), *boils* etc.—These are due to the infiltration of the larvæ of hook worms into the skin (Rogers and Megaw *loc cit*). Some authorities like Savill say that urticaria is also caused by the worms. During the rains tea estate labourers frequently come to hospital for treatment of these troubles.

Leucoderma (vitiligo)—This also has been observed in some cases. In the acquired variety of the condition some chronic bowel disturbances are common (Majumdar, *loc cit*), and the intestinal parasites usually give rise to such bowel disturbances.

NERVOUS SYSTEM

Distemper and restlessness—It is a common experience of the clinicians that the children suffering long from worm infestations become very fretful and restless. This may happen either due to instability of their nervous system which is easily disturbed even by minor troubles, or toxæmia caused by the toxins of the worms, or both. Intense itching of the anus produced by the thread-worm may also cause fidgettiness in children.

Apathy and melancholia—In some cases of ankylostomiasis the patient may develop also these mental disorders (Savill, *loc cit*).

Delirium, grinding of the teeth at night, picking at the nose and night terrors—These reflex phenomena are commonly seen among the children with worm infestations.

Convulsion—This is undoubtedly a very common emergency in children. Intestinal worms are often held responsible for the condition. Athala (1943) says that regular convulsions may closely resemble epileptic fits in children. The worms can give rise to toxæmia as well as to an irritative condition of the alimentary canal, so both the toxic and reflex causes may be responsible for the convulsions in worm-infected children. Also coma may be associated with ascariasis in children (Majumdar, *loc cit*).

Headache—In a few individuals obstinate headache has been found to have accidentally disappeared after anthelmintic treatment. Hepatic or gastric derangements wrought by the vermes may be attended by the symptom. Amelioration of pain in such cases can be partly ascribed also to the purgatives that are generally administered with the vermicide.

Defective vision—According to Savill amblyopia may occur in ankylostomiasis. The author has come across many cases of night-blindness (nyctalopia) among the tea-garden labourers who are usually good harbourers of various worms. Avitaminosis was also present in some of the cases.

Fever—Worm infections are often associated with pyrexia of different types of varying degrees. Short convulsive fever is often found in children. Ascariasis may simulate typhoid fever and also malaria in some cases (Debsarma, 1945) and thus may mislead the clinician. Of course these diseases can be excluded by the absence of their other cardinal characters. This constitutional symptom is caused probably by some pyrogenetic toxins secreted by the worms in the intestines.

CONCLUSION

This paper is in no way complete, it has presented only a few observations made by different authors on the subject, together with a hint of a very few points raked from the author's meagre experience. However, it is obvious from the above that intestinal parasites can create a great deal of trouble to human life and some of their effects on the system are undoubtedly deleterious. Babies and young children suffer most from their infestations as they have not yet acquired any immunity towards the worms. Even babies three months old have been known to pass ascaris eggs.
(Concluded at foot of page 11)

INTESTINAL HELMINTHIASIS

D N DEBSARMA, M B,
Koilamari Tea Estate Hospital, Lakhimpur, Assam

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B P KACKER,

*From the Department of Children's Diseases,
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In children the temperature is more often of an intermittent type, may be remittent, and the classical continued type of temperature is less frequently seen, whereas in adults the temperature is usually of a continued type. It is known that in a large bulk of adult patients the temperature becomes intermittent in character towards the end of the disease which undoubtedly is due to the gradual development of immunity against the infection. Arguing from this observation the authors opine that the presence of intermittent temperature in children coupled with the mild course of the disease point to the presence of a bigger dosage of immunity in them against typhoid fever. And this immunity must necessarily be natural and not acquired. Another point has been noted by the authors that leucopenia is not common in children and this is in marked contradistinction to that observed in adults. Children usually run an uncomplicated course.

Below are recorded three cases of typhoid fever in children which will be of value in establishing the new features discussed above. Cases No 1 and 3 were referred to us along with their temperature charts from Tundla by Dr G P Neoga, L.R.C.P. (England).

Case 1—J P, 5 years, Hindu male child. Complaint—fever, duration 18 days.

Physical examination—Temperature 102°F, pulse 120 and respiration 20 per minute. Appearance completely non-toxic. Tongue coated. Abdomen, heart, lungs and other systems normal.

Investigations—Blood total leucocytes 8,900, polymorphs 38, lymphocytes 50, mononuclears 12. Widal test positive 1 in 250 against typhoid.

Points of interest—1 Completely non-toxic appearance. 2 Absence of leucopenia. 3 Presence of intermittent fever (*vide* Temperature Chart I). 4 Uncomplicated course. 5 Rapid convalescence.

Case 2—K S, 10 years, Hindu male child. Complaint—Fever, duration 10 days.

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Points of interest—1 Completely non-toxic appearance. 2 Absence of leucopenia and eosinopenia. 3 Presence of remittent fever with about 3 degrees fluctuation (*vide* Temperature Chart II). 4 No complications. 5 During the whole course the child used to feel very hungry. 6 Presence of relative bradycardia during second week. 7 During convalescence there was still further bradycardia—pulse rate 58 per minute.

Case 3—S, 4½ years, Hindu male child. Complaint—Fever, duration 62 days.

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Points of interest—1 Presence of intermittent fever during the whole course of the illness (*vide* Temperature Chart III—whole chart not reproduced). 2 The diagnosis of typhoid fever was clinical. Widal persisted to be negative. This was no doubt due to the absence of the development of agglutinins in the blood. This may explain such a long course. 3 Uncomplicated course. 4 Absence of marked leucopenia.

GLUCOSE TREATMENT OF DIABETES AND EFFECT OF SEDATIVES WITH A CASE REPORT

B. C ROY, M.D., D.S.C., M.R.C.P., F.R.C.S.,
H. N. MUKHERJEE, B.S.C., M.B., D.I.C. (LOND)

AND

D. M. CHAKRAVARTY, M.B.

Biochemical Department, Sir Nilratan Sircar Research
Institute, Carmichael Medical College, Calcutta

The view that the hyperglycaemia of diabetes is not really a pathological manifestation but a physiological response was first put forward by Roy and Mukherjee (1935, 1939). The effect of glucose given intravenously to diabetics is usually beneficial. Thus Roy and Mukherjee (1935) found "that glucose when given intravenously in diabetes was utilised. The introduction of glucose in adequate amounts intravenously quickly brings available glucose to tissues and liver and thus stops hepatic gluconeogenesis and reduces acidosis and ketosis. After a few days in spite of receiving daily injections of 150 c.c. of 25 per cent glucose intravenously the hyperglycaemia was definitely reduced."

The effect of intravenous glucose therapy in a case of diabetes is reported here.

CASE REPORT

History—P. G., Hindu, male, age 46, was admitted to this Hospital on 8-6-46.

Complaints—Frequency of micturition and polyphagia for last 3 years and glycosuria during this period. Left eye has got no vision due to glaucoma and there is a cataract in his right eye for last 3 years.

Systemic Examination—Liver 1" palpable below costal margin. A moderately large hydrocele in the right side of the scrotum of eight years' standing.

The patient attended the outdoor of the Carmichael Medical College Hospitals on 23-4-46 when his urine was examined qualitatively and sugar was found in the urine. He could not be then admitted in the Hospital but was put on a restricted diet. As mentioned above he was admitted to this Hospital on 8-6-46. Body weight of patient was 106 lbs.

Treatment—The patient was kept on a restricted diet of 4 chapattis (510 calories), 8 oz. of boiled vegetables (80 calories), and 8 oz. of milk (144 calories), morning and evening. He was thus kept on a diet of 1468 calories per day. His urine was sugar-free. So a sugar tolerance test was done on 24-6-46 after administering 50 gms of glucose by mouth.

Initial fasting blood sugar	0.077%
Blood sugar after ½ hr	0.133%
" after 1 hr	0.143%
" after 1½ hr	0.167%
" after 2 hr	0.182%

The test showed that though his fasting blood sugar level was normal his glucose tolerance was poor and definitely like a diabetic.

Intravenous Glucose Therapy—He was then given 50 c.c. of 25 per cent glucose solution intravenously twice daily from 25-6-46 to 1-7-46.

A glucose tolerance test was done on 2-7-46 with 50 gms of glucose by mouth.

Fasting blood sugar	0.063%
Blood sugar after 1½ hr	0.118%
" after 2 hr	0.133%

The diabetogenic function of the anterior pituitary is now recognised by physiologists. Opium derivatives are used empirically by practitioners of indigenous medicine in diabetes. To ascertain whether opium has got any depressing effect on the anterior pituitary therapy alleviating the diabetic condition the patient was given 15 minims of liq. morphine hydrochlor thrice daily by mouth along with the intravenous glucose treatment from 3-7-46 to 10-7-46.

A sugar tolerance test was again done on 11-7-46.

Fasting blood sugar	0.095%
Blood sugar after 1½ hr	0.105%
" after 2 hr	0.111%
" after 2½ hr	0.118%

From this sugar tolerance test it appears that morphine may have a beneficial effect.

He was next given methophenobarbitone (Rutonal—May & Baker) ½ gr. in the morning, ½ gr. at noon and 1½ gr. at bed time by mouth along with 50 c.c. of 25 per cent glucose solution intravenously twice daily from 12-7-46 to 17-7-46.

On 18-7-46 again a sugar tolerance test was done.

Fasting blood sugar	0.069%
Blood sugar after 1½ hr	0.125%
" after 2 hr	0.105%
" after 2½ hr	0.069%

It appears that methophenobarbitone (Rutonal—May & Baker) has an inhibitory action on the anterior pituitary thereby improving the glucose tolerance. But further experimental evidence is necessary.

Finally the patient was kept on the restricted diet alone and intravenous glucose therapy was stopped from 19-7-46 to 29-7-46.

On 30-7-46 a final sugar tolerance test was done.

Fasting blood sugar	0.08%
Blood sugar after 1 hr	0.083%
" after 1½ hr	0.095%
" after 2 hr	0.091%

It should be noted that the patient was kept on the measured restricted diet mentioned above throughout the period of investigation, i.e., upto 30-7-46 and he was given 50 c.c. of 25 per cent glucose solution intravenously twice daily from 25-6-46 to 17-7-46.

COMMENTS

Though the patient on admission in the Hospital did not pass sugar in the urine and his fasting blood sugar level was normal his glucose tolerance was like that of a diabetic. After the first period of glucose therapy, i.e., from 25-6-46 to 1-7-46 (a period of eight days) the patient showed an improvement in his glucose tolerance. The 2nd test period (3-7-46 to

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and the ureter freed into the iliac region, the wound was stitched up, leaving the kidney lying out of its lower end. A second muscle-splitting incision having been made in the left groin, and the peritoneal cavity opened, a large mass of tuberculous glands lying in the mesentery of the small intestine was removed. The peritoneum

was then cut through on the back wall and the pelvic ureter exposed. As it was traced over the pelvic brim it was dilated and buried in dense iron-like adhesions, but just above the bladder it became narrowed and stenosed. Here it was cut across between ligatures, the wound stitched up with drainage, and the whole specimen removed from behind.

Convalescence was uneventful and the urine became sterile and free from pus within three weeks.

Specimen (Fig. 28).—The ureter is seen tied off at its junction with the bladder. Above this the ureter is dilated and tortuous and the kidney hollowed and injected.

Case 8.—Acquired stricture of left ureter. Infected hydro-ureter and hollowed kidney.

A married woman, age 36, examined in October, 1926, stated that in February, 1915, she got soaked when in evening dress, and was seized with fever and pain in the left side of the back. On and off ever since she has had these attacks at intervals of about a year: high fever, back-ache, increased frequency, and strangury. Between the attacks the bladder is usually troublesome. She has had courses of vaccines and drug treatment without relief. There was no X-ray evidence of stone. The urine drawn off by catheter was hazy with pus and the colon bacillus. The bladder, which would only hold 4 oz., showed marked basal cystitis. The right kidney proved healthy and efficient. A No. 5 ureteric catheter would not pass more than one inch up the left ureter. A fine left ureteric catheter was passed to the kidney; a specimen was taken which contained pus and *B. coli* and was functionally of poor value. A pyclogram showed the catheter curled up in a large hydro-ureter, and above this a small kidney with hollowed calices and dilated pelvis.

OPERATION.—On March 9 the left kidney was exposed from behind, the pedicle isolated and ligatured with catgut. The kidney was not enlarged or lobulated. The pelvis was not much distended, but the ureter was dilated, thin-walled, and bound down by tough adhesions, the result of inflammation. It was separated as far as the iliac crest, and the wound stitched up with the kidney hanging out of the wound.

The patient was then turned on her back, the left rectus turned outwards, the peritoneum not opened but pushed away from the left lateral wall of the pelvis, and the ureter identified. The

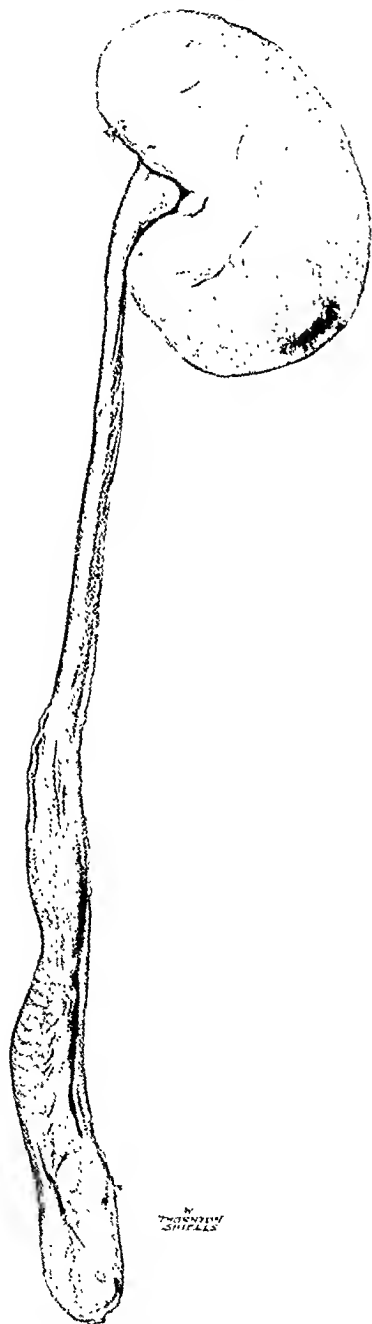


FIG. 29.—Case 8. Acquired stricture of left ureter. Infected hydro-ureter and hollowed kidney.

ureter was far larger in the pelvis than it was in the loin, tortuous, and with tough fibrous bands full of veins and blood-vessels very adherent to the peritoneum. The uterine arteries and veins, spread out fanwise across the ureter in the fibro-fatty tissue in which they were buried, had to be separated carefully and ligatured in series. With patience this was gradually accomplished, and the outer side of the ureter was cleared as far as the bladder, but the inner attachment proved more difficult to clear, and just at the lower end the peritoneum was torn. Eventually the ureter was cleared right down to the wall of the bladder. The last inch was quite narrow as it entered the bladder. Immediately above that the ureter was widely dilated and its wall thickened. It gave the impression that the large artery and veins running downwards from the uterine vessels to the side wall of the bladder and top of the vagina had compressed the lower end of the ureter and so caused the obstruction. Possibly there had been inflammation in the parametrium on the left side around these vessels which had formed constricting bands of fibrous tissue around the blood-vessels. The ureter was cut across between ligatures at the entrance to the bladder, and the whole specimen was removed entire through the loin. The wound was stitched up with drainage. Recovery was uneventful.

Specimen (Fig. 29).—The whole of the pelvic ureter above the ligature is seen very much dilated and its walls thickened. There is atrophy with hollowing of the infected kidney.

Case 9.—Congenital defective kidney and ureter. Infected hydro-ureter and hydronephrosis without an organic obstruction in the ureter.

A girl, age 10, first examined in September, 1926. It was stated that she suffered from imperative micturition and constant bed-wetting. There was no pain and very little effect on the general health. The urine contained a quantity of pus and *B. coli*. The left kidney was natural and efficient. The right kidney was heavily infected. The right ureter was completely patulous, so that pyelographic media injected into the bladder flowed freely into the right ureter and kidney. Pyelograms showed a wide tortuous hydro-ureter and a dilated kidney pelvis with a small relic of kidney substance lying on the top of it. For some months efforts were made to disinfect the kidney and ureter by filling up the bladder with antiseptics and placing the child on a bed with her head down and feet up. The infection somewhat abated, but as the symptoms were in no way relieved it was decided to operate in June, 1927. The right kidney was exposed from behind and isolated, the ureter was then exposed through a paramedian incision, traced as far as the bladder wall, and removed in one piece with the kidney. Recovery was uneventful.

Specimen (Fig. 30).—This consists of a small lobulated relic of kidney substance about the size of a walnut, lying at the top of a dilated and thick-walled infected pelvis and ureter. The ureter was completely patulous at its entrance into the bladder.

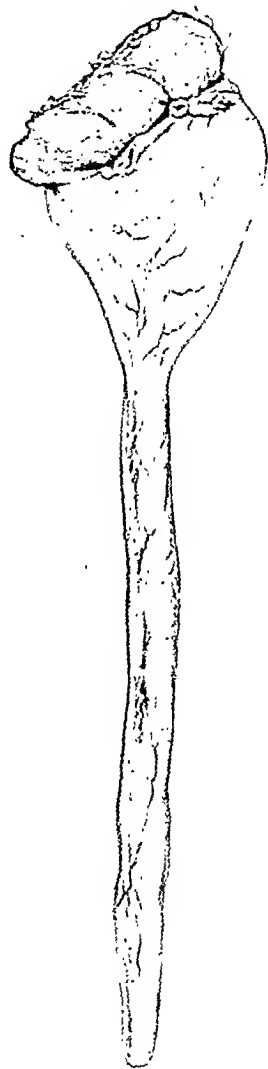


FIG. 30.—Case 9.
Congenital defective kidney and ureter. Infected hydro-ureter and hydronephrosis without organic obstruction in the ureter.

hydro-ureter, the causation of which is obscure. There appears to be no organic obstruction to the ureter or kidney in the lumen, in the wall, or without the wall of the ureter. The condition, which may be unilateral or bilateral, may be explained on the following hypothesis. The subject is born with a congenital defect of the ureter, just as a subject can be born with a congenital club-foot. This defect resides in the wall of the ureter, which is born so wide that the muscle coat of the ureter is from birth unable to contract so as to close the wall of the ureter in peristalsis and to close the normal valvular opening of the ureter into the bladder which exists in normal persons, but which does not exist in these defective subjects. At every act of micturition the bladder closes down in the urine in the bladder, subjecting it to a certain pressure. This pressure results not only in emptying the bladder by the urethra, but also forces urine backwards and upwards into the patulous ureter and kidney pelvis. The muscle coat of the defective kidney pelvis and ureter undergoes hypertrophy in an attempt to resist the pressure of the bladder muscle. This resistance is, however, ineffective, and gradually the kidney substance becomes hollowed out and destroyed from the back-pressure of the bladder muscle at each act of micturition. A very small increase in the pressure in the renal pelvis is sufficient when acting over many years to cause back-pressure atrophy of the renal parenchyma. For instance, in prostaties with giant overflow bladders, experience shows that on balancing the renal pressure after suprapubic cystotomy against the gravity of a column of water, this pressure is seldom more than six inches to at most a foot of water.

In this way it is possible to suggest an explanation of these cases of hydro-ureter and hydronephrosis with no organic obstruction, which are not uncommon, and resemble Hirschsprung's disease of the colon or congenital megacolon.

Case 10.—Double kidney with double ureters, one of which opened into the bladder, the other into the vagina.

A single woman, age 32, first examined in October, 1925, stated that in October, 1923, she had an acute attack of pyelitis of the right kidney, followed by further attacks in November, 1924, and in March and July, 1925.

The patient looked wasted and poisoned. The urine contained pus, the colon bacillus, and streptococci. Pyelograms and functional tests applied showed a healthy left kidney, but on the right side a small infected, hollowed kidney.

OPERATION.—The right kidney was explored from behind at an operation in December, 1925. A small narrow ureter was found running up to the renal pelvis. On the inner side of this was a thick-walled hollow structure, in appearance like a dilated ureter, which ran to the extreme upper pole of the kidney to end blindly in a small nodule of fibrous tissue. The renal artery and vein, which coursed in front of the double ureters, were ligatured with catgut, the kidney was pulled downwards and outwards, and the lower portions of the double ureter were defined as far as the iliac crest. Here the two ureters were divided between ligatures, and the wound was stitched up without drainage.

On investigating the specimen removed (*Fig. 31*) it was found that the lower outer ureter ran to a moderately dilated renal pelvis, as shown in the pyelogram, which pelvis belonged to the lower half of a double kidney. The upper inner ureter, dilated and thick-walled, ran to the extreme upper portion of the kidney, where it ended in a small nodule of tough fibrocytic tissue. This tissue was found to be an atrophied bud representing the upper half of a double kidney.



FIG. 31.—Case 10. Double kidney (A) with double ureters (B), one of which opened into the bladder, the other into the vagina.

During convalescence it was discovered that the patient suffered from an excessive purulent vaginal discharge, which apparently had been present ever since she had had measles in 1917, and which she had never previously mentioned to anyone. On further questioning, she acknowledged that when young she was aware that urine used to leak from the vagina. In November, under anaesthesia, a long thickened crest could be felt running along the right side of the vagina towards the right lateral fornix. Here a tiny opening could be seen from which large quantities of foul, greenish, mucopurulent fluid were exuding.

SECOND OPERATION.—In January, 1926, a second operation was performed. The right rectus was turned outwards and the peritoneal cavity opened. The abnormal ureter could be seen lying behind the peritoneum which covered the iliac fossa, tortuous and swollen with mucus. The posterior peritoneum was incised from the caecum down to the broad ligament,



exposing the double ureters, which were deeply buried in dense fibro-fatty adhesions full of veins. The upper ends ligatured at the first operation, buried in a mass of retroperitoneal fat, were freed and brought downwards and forwards into the pelvis until the uterine vessels were reached, crossing from without inwards and over the ureter, and were cut across between ligatures. The small outer ureter was found to pass across the front of the upper dilated ureter and run forwards through the broad ligament to the back of the bladder. This was separated and cut across between ligatures. The dilated upper ureter was traced outwards to the top of the right lateral vaginal wall, where it was surrounded with dense fibrous bands enclosing veins and arteries, which made dissection very difficult. At this point it divided into two branches. The inner one communicated through a fine opening into the right lateral fornix with the vagina itself. The outer one ran downwards and outwards for some inches as a blind pouch along the lateral wall of the vagina, corresponding to the ridge previously noticed in the vaginal wall. A clamp was then placed as low down as possible, and the two branches of the ureter were cut across below the clamp. When this was done a certain amount of purulent fluid escaped from the blind pouch still attached to the vagina. This cut left a wide hole leading from the upper end of the blind pouch along the lateral wall of the vagina into the upper end of the vagina itself. A tube was inserted through the hole into the vagina and left to drain the space opened up in the retroperitoneal tissues. The cut in the posterior peritoneum having been stitched up and the appendix removed, the wound in the abdominal wall was stitched up as usual. The wound healed satisfactorily, but the patient continued to suffer from occasional attacks of dull pain in the pelvis, always reduced by a sudden discharge of pus from the vagina.

At a further examination under anæsthesia in May, 1926, a small cystic swelling could be felt on the right side of the uterus which opened into the right side of the fornix and was continuous with the thickened crest which ran from the opening downwards and outwards along the right wall of the vagina. This crest and as much as possible of what remained of the blind pouch were cut away through the vaginal wall with scissors.

Since then the patient has gone on well, and has been able to hunt and live an active life, although she occasionally gets mild attacks of pelvic pain relieved by the passage of a small quantity of mucus from the vagina.

Specimens (Fig. 31).—

A. Consists of a double kidney with two ureters. The upper kidney is represented by a small fibrous nodule lying at the top of a wide, thick-walled ureter, heavily infected.

B. This consists of the lower portion of the two ureters. The small lower ureter crosses the upper ureter in front from without inwards and ends in a ligature at the point where it entered the bladder.

The upper ureter is widely dilated and the wall thickened by inflammation. It runs downwards and outwards and is seen to end in two branches: the inner one is small and terminates by an opening into the top of the vagina, the outer one is larger and was continued in life some three inches farther down to end as a blind pouch lying in a thick-walled crest in the right lateral wall of the vagina. This pouch, not shown in the drawing, was removed at the third operation.

The full diagnosis of this case was only possible at operation, especially as the patient had concealed a most important part of her history till after the first operation.

Case 11.—Congenital cystic kidney with ureter opening into the vagina.

A child of 12 years, first examined in 1913. It was stated that since birth her mother had noticed large quantities of pale urine leaking continuously from the vagina. An opening could be seen in the left lateral fornix of the vagina, from which pale urine containing 0.2 per cent urea exuded.

OPERATION.—The left rectus was turned outwards. A small cystic kidney was found on the left side, and the ureter could be traced down from this to the top of

the vagina. The kidney was freed from its pedicle, and the ureter cleared to the top of the vagina and cut across. The kidney and ureter were then removed in one piece. Convalescence was uneventful, and there was no further trouble.

Case 12.—Congenital ectopic pelvic kidney complicated with calculus and infection.

A woman, age 44, examined in April, 1927, stated that for sixteen years she had suffered from intermittent attacks of fever, bladder strangury, and pyuria. Recently she had suffered from severe attacks of pain in the front and back of the left loin, with nausea and faintness. X-ray examination revealed a curious group of shadows shaped like gall-stones lying in the middle of the pelvis. Pyclography revealed a healthy right kidney lying in the normal position, and an infected ectopic hydronephrotic left kidney of poor functional value lying in the pelvis and containing stones.

OPERATION.—In May, 1927, the left rectus was turned outwards from the middle line, the peritoneum opened, and the intestines cleared out of the pelvis. The left

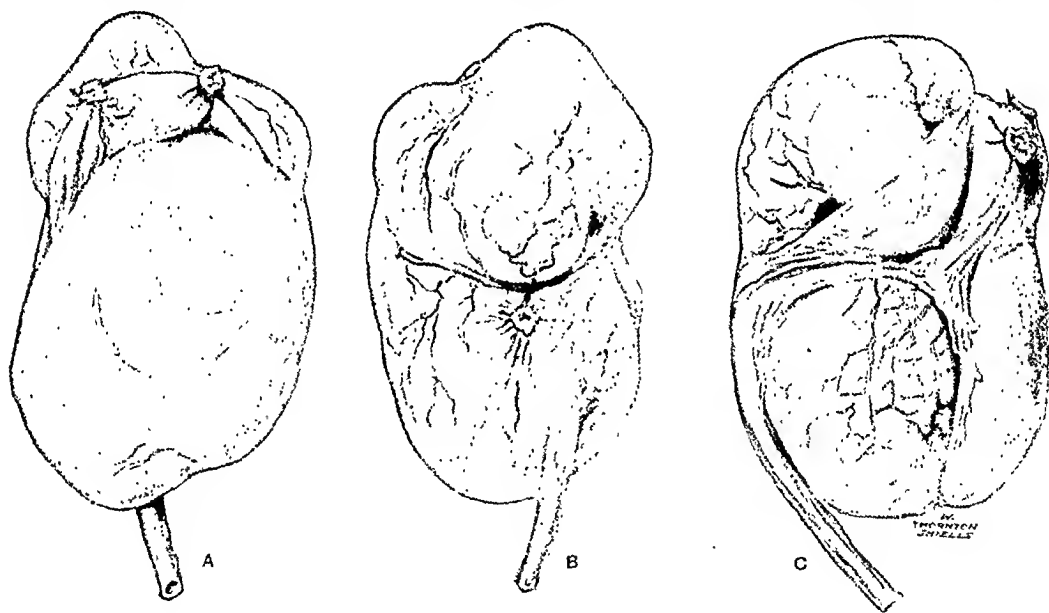


FIG. 32.—Case 12. Congenital ectopic pelvic kidney complicated with calculus and infection. A, Front view; B, Back view; C, Side view.

kidney was lying behind the peritoneum below the brim of the pelvis. The kidney itself lay on the left side of the dilated pelvis, from which the ureter passed downwards and backwards behind the kidney to penetrate the left broad ligament to reach the bladder. The kidney was supplied by three sets of blood-vessels. There were two arteries and two large veins running down from the front of the aorta and vena cava over the bifurcation of the aorta to enter the upper end of the kidney and suspend it. Large veins emerged from the main veins to distribute themselves in the perinephric fat. These had to be tied in series, the peritoneum being cut through and the kidney and pelvis gradually cleared. The two upper sets of blood-vessels were ligatured with catgut and the vascular pedicle was freed. The ureter was then traced down through the broad ligament to the wall of the bladder. There were two or three large arteries and veins running up in a plexus from the uterine artery and vein, surrounding the ureter and passing up to the kidney substance. These had to be ligatured in turn, and finally the ureter was left attached to the wall of the bladder free from blood-vessels. The ureter was tied off close

to the bladder wall and the whole specimen removed. The peritoneum having been stitelled up on the back wall with catgut, the abdominal wound was united without drainage in the usual manner. Recovery was uneventful.

Specimen (Fig. 32).—

A. The kidney is viewed from the front exactly as it lay in the body. The kidney substance is below and to the left. Above there can be seen the ligatures round the two upper groups of blood-vessels, between and below which is a large venous sinus. Above the ligature can be seen a portion of the dilated kidney pelvis.

B. The kidney viewed from behind and hidden by the grossly distended renal pelvis. The pelvis is constricted around its middle by a wide band of fibrous tissue containing a large vein which forms another venous sinus connecting the lateral veins on each side. The ureter comes off underneath this band and vein, and is constricted by them so as to possess a valvular opening.

C. This shows a lateral view of the specimen. The kidney is in front, the dilated pelvis behind. The origin and constriction of the ureter by the band and vein are made clear.

Fig. 33 represents the main blood-supply of the organ from three different sources, and indicates the stones lying loose in the renal pelvis. In addition to the main blood-vessels and their anastomoses as shown, there was a set of six large anastomosing veins running in front of the specimen in the perinephric fat which had to be tied off and removed before it was possible to expose the kidney itself.

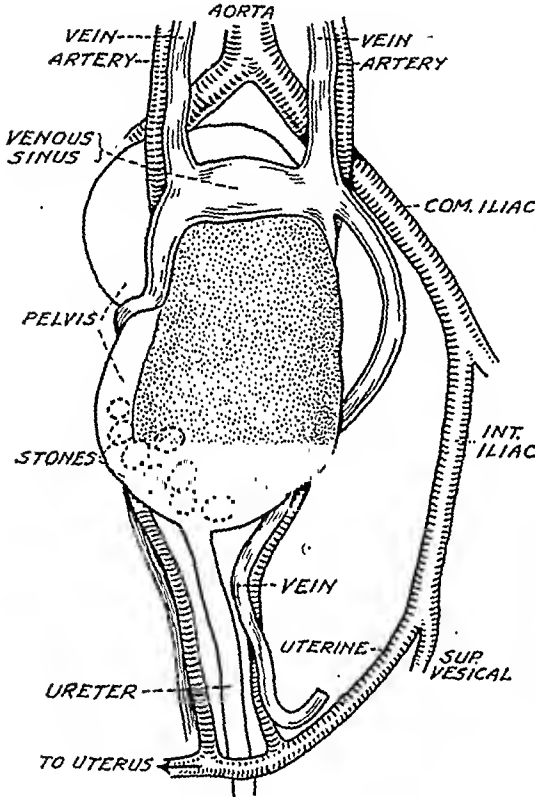


FIG. 33.—Case 12. Diagram to show the blood-supply of the kidney in Fig. 32.

The extraordinary wealth of the blood-supply of the pelvic kidney is a feature met with in pelvic ectopic kidneys, and has been known to deter operators from proceeding to nephrectomy in such cases. The exercise of gentleness and patience is required to surmount this difficulty.

Though the kidney was lying in the pelvis, yet the renal pain experienced by the patient was in the left loin in the usual position of renal pain. The writer knows of three similar cases operated on by others in which the same feature as regards the position of the pain was noticed. This fact makes the diagnosis all the more difficult.

REFERENCES.

- ¹ HYMAN, *Ann. of Surg.*, 1923, Sept., 387.
- ² LILIENTHAL, *Ann. of Surg.*, 1911, April.
- ³ LATCHAM, RAYMOND, *Jour. of Urol.*, 1922, viii, 257.
- ⁴ KIDD, FRANK, *Lancet*, 1913, June 7.

THE EARLY DEVELOPMENT OF HOSPITALS (BEFORE 1348.)

*(Being the Thomas Vicary Lecture delivered at the Royal College of Surgeons of England,
on November 3, 1927.)*

BY GEORGE PARKER,

CONSULTING PHYSICIAN TO THE BRISTOL GENERAL HOSPITAL.

I DESIRE in the first place to express my sense of the honour of being invited to address the Royal College, and also my pleasure at seeing here to-day representatives of the Barbers' Company who endowed this lecture. It is a striking thing to reflect that 500 years ago they were taking their share in founding surgical lectures—just as they are doing to-day.

In the twelfth and thirteenth centuries of our era, a remarkable enthusiasm for creating hospitals came to a head. It had grown up gradually, and spread over many distant countries, from Burma, Siam, and Ceylon, to Syria, Persia, Egypt, and the whole of Western Europe, excepting perhaps Russia. I propose to inquire what were the sources of this movement, and how it was that hospitals had grown up everywhere in that huge area.

Now an institution or idea may arise in the progress of civilization in two or more distant places simultaneously, and there has been much discussion whether such things really have a common origin, or start independently. Thus, did the institution known as a hospital start from a single source, and the idea of its utility spring from a single mind? The earliest beginnings we know of can be traced to about the sixth century B.C., in places far apart both in the West and distant East, and hospitals do not appear in earlier times or outside a certain group of countries, with the one exception of Mexico. Thus there is no evidence of them in Assyria, Babylon, early Egypt, or China, in spite of the medical skill and civilization there existing.

The idea once started, however, appealed to various religions and to various rulers of men. The Buddhist or Christian ascetic, who neglected his own body, still toiled to build hospitals for the comfort of the sick. Among founders were many blood-stained Indian, Roman, or Moslem princes, Greek city oligarchies or tyrants, half-savage barons, the Kaiser Karl the Great, Haroun Ar Raschid, and our own Athelstan. In Western Europe, especially in the twelfth and thirteenth centuries, we find a host of private persons and societies competing with their rulers in the work. Two or three records will give some idea of the results at that date. Thus in France in 1226 Louis VIII left legacies to 2000 hospitals and to 200 poor-houses. In England, besides monastic infirmaries, 577 hospitals and asylums were founded between 1100 and 1400. In Germany and Italy the numbers were huge; one society alone is credited with building 900 in the thirteenth century. In Ceylon and Burma they seem to have been ubiquitous, and probably, too, in the shrunken Byzantine Empire. In the great Moslem world at least twenty cities boasted of

having one or several hospitals each, some of them being the most perfect of that age. The enthusiasm only faded about the time when the Black Death and the invasions of the Mongols and other barbarians wiped out a large part of the builders and the patients. We may add that it was revived suddenly, at least in Western Europe, in the eighteenth and nineteenth centuries, but in the history of the idea such ebbs and flows had occurred before.

Let us pause to consider what is meant by a hospital. In the strict sense it is a place designed for the residence under favourable conditions of the sick or wounded during treatment, or during the progress of a self-limited disease. The better class of hospitals, too, provides for the observation and registration of symptoms, for baths, surgical theatres and instruments, laboratories, and other facilities for treatment. Many, too, provide for the training of students, surgeons, and physicians.

We must distinguish them from (1) rest-houses for travellers, (2) orphanages, and, (3) homes for the poor and aged. On the other hand, asylums for the insane or for lepers, or consumptive sanatoria, may be either true hospitals or merely homes for segregating affected persons, in fact quarantine stations. Again, out-patient dispensaries present some difficulties, since elaborate treatment, as in many eye clinics to-day, researches, and the training of students may be carried on in them; but, if we are to regard them as hospitals, how about the private office or surgery of a practitioner? They are all institutions for the cure of disease, and the question of ownership is unimportant. However, ordinary usage tends to restrict the term hospital to places with beds for the patients, and I think quite rightly. Many ancient institutions combined true hospitals with other departments under the same roof, such as orphanages and asylums for the poor and insane. The mediæval hospital was usually like our Poor Law infirmaries, where both acute and chronic cases are taken in, nursed, and treated. It is not always possible to say what kind of institution historical records refer to, though Justinian carefully separates nosocomia from poor-houses, rest-houses, orphanages, and homes for aged persons or foundlings. I shall endeavour to restrict my remarks to what were true hospitals wholly or in one department, though a hard-and-fast line is impossible. It is astonishing how perfect in every detail some of the very ancient ones were, but as to others we know nothing but a single word in a charter or inscription.

Let us now take up the story of the rise of hospitals in the various countries.

THE BUDDHIST HOSPITALS.

It is not clear to me that any sprang up in the life of Gautama; but after his death in 543 B.C. monasteries very quickly arose throughout the Buddhist world, each containing an infirmary open to any sick man, or, indeed, to animals. These hospitals, formed to carry out the precepts of the religion, existed then in great numbers from 500 or 543 B.C. We have no evidence that the idea was taken from any previous institutions, either Indian, *Persian* under the first Achæmenians, or from an early Indo-Mediterranean world. They were clearly prior to the Grecian influences of Alexander's invasion, and at present we can only regard them as a new idea. In the *Arthaśastra*,

book ii, cap. iv, possibly written by Kautilya under Chandragupta about 325 B.C., we find hospitals spoken of as a necessary thing in fortified towns. These would probably be military hospitals, as distinct from the religious foundations. In the second Edict of Asoka about 257 B.C. we read :¹ "Everywhere in the dominions of his Sacred and Gracious Majesty, and among his frontagers as far as Antiochos [theos] the Greek king, and the neighbours of that Greek king, have healing establishments for men and animals been set up and medical herbs have been imported or planted." Thus he claimed that not only in India, but also in Syria and Mesopotamia, had such Buddhist institutions been founded. However, no trace of them has since been discovered in those western lands. Let me recall to you a few of the other well-known royal foundations. King Gamani in Ceylon, B.C. 161, claimed that he had maintained hospitals in 18 places. King Buddhadasa in 341 A.D. not only provided hospitals for men and animals, but practised surgery himself.² Fahien, the Chinese pilgrim, 405-11 A.D., describes the excellent free hospital supported by benevolent chieftains at Pataliputra ;³ all the sick from the provinces near came there, were attended by a physician, but stayed only so long as they were ill. King Harsha, after 30 years of warfare, about 648 A.D., established hospitals throughout his empire in imitation of Asoka. Huen Tsien, another Chinese, tells us that these 'houses of benevolence' existed in the Punjab, in Central India, and in two other provinces. Recent opinion places in this Buddhist period the really able surgeons Charaka and Susruta, men who, as is well known, practised laparotomy, suture of the intestines, and rhinoplasty. Still later on, in 1190, Parakrama in Ceylon is found hospital building, one being mentioned of great size and splendour.

Thus from before 500 B.C. downwards Buddhist hospitals were perpetually being built (1) by victorious rulers, (2) by monasteries, or (3) by individuals. Where Buddhism died out, as in India proper, they vanished, except at Surat and a few other places, but they still exist to-day in Burma, Siam, and Ceylon.

ZOROASTRIAN HOSPITALS.

Of Zoroastrian early hospitals very little is known, though we might expect to find them under that religion. It has been said, indeed, that a great part of the Persian records were destroyed by two conquerors, Alexander when drunken, and the teetotal Arabs under Omar. However, the *Avesta* and a great deal more survived, but there is so little recorded on the subject, that it has been thought that Persian medicine was really negligible, and in support of this it is pointed out the kings were so keen on getting Greek doctors for their courts that they kidnapped them when they could not hire them. Still, in the *Dinkard*, which seems to be an abstract of earlier liturgies, it is laid down as one of the duties of kings to found hospitals in important centres and to provide physicians and drugs.⁴ We do not know, indeed, how far the kings in the humane and well-organized Persian Empire carried out this rule until we come to the important embassy sent by Chosroes I or Khusraw to India about 550 A.D. to study the Buddhist medical system, and to bring back drugs to Gondishapor. Here was a flourishing medical school composed partly of Zoroastrians and partly of Nestorian Christians, and soon a great hospital

appears there also. Dr. Bargoia's mission to India found the Buddhist hospitals in great activity, and he was able to bring over teachers, drugs, and many things which greatly impressed his contemporaries. Thus, it is claimed that the game of chess, and certain popular Indian tales, the Fables of Pilpay, were brought by him, and, curiously enough, among the drugs which were first introduced there into the Western world about that time was cane sugar.

This hospital and school of Gondishapur combined then Buddhist, Greek, and Zoroastrian teachings, and it flourished vigorously. Its influence spread far and wide, and in later times notably among the Moslems. For many generations it was the chief source of learning in the East, as Clifford Allbutt remarks.

HOSPITALS IN THE WESTERN WORLD.

These originally started from two sources, the Æsculapian cult on the one hand, and on the other a provision for sick citizens devised by the practical Greeks, who were intent on forming a model city state, and had a large body of able medical men at their command.

1. *The Temples of Asklepios*, beginning perhaps with Epidauros about 500 B.C., claimed many cures, which in early times were said to be produced by the direct action of the Numen, but in later days generally through remedies prescribed by the god to the patient while sleeping in the shrine. Hence the term incubation. The remedies included baths, ointments, bleeding, massage, operations, and drugs. Fees were certainly charged to the well-to-do, and apparently beds were provided for patients while the remedies suggested were being applied. It is estimated that there were over 320 of these temples, and of these nearly 200 existed before the time of Alexander the Great. There were also similar healing shrines of Pluto, Amphiarios, Dionysos, and, from the time of the first Ptolemy, others of Isis and Serapis, the latter being a somewhat doubtful divinity who took the place of older Egyptian gods such as Ptah and Imotep. The relation of Greek and Egyptian incubation shrines is most obscure, but I know of no evidence showing that the Egyptian were earlier. To show how widely the incubation cult had spread, we may mention that Arrian speaks of seven of the companions of Alexander as sleeping in the temple of Serapis at Babylon to inquire as to their leader's illness. He has been criticized for this statement, but it seems that the temple was really one of Baal Zipur, who was later on identified with Serapis.⁵

Now Cos was originally an Asklepiian shrine, and in later times it possessed a magnificent hydropathic establishment or hospital, the ruins of which still exist. Possibly this hospital was of a different origin, and due to the great medical school which had grown up there from the time of Hippocrates; and this brings us to the second class.

2. *Public Health Officers*.—Side by side with the temple shrines were great numbers of private secular doctors carrying on their art, and also a highly-paid and well-trained public medical service even as early as 600 B.C. Democles, for instance, a little later, about 500 B.C., is found drawing a salary equal to £250 and £500 per annum as city physician. These public medical officers appear not only in all cities of Greece itself in the fifth or sixth century, but also in Sicily and Southern Italy, and can be traced from the time of Plato

down to Roman times everywhere. Hence we can, I think, very well accept the conclusion reached by our late friend and teacher, Clifford Allbutt, that there was public provision universally for the sick citizen, first in Greek cities and later on in the Roman Empire.⁶ Now there were not only physicians in all Greek cities. There were hospitals as well, for all doctors, public and private, had *iatreia* or dispensaries, and in some of these were beds for in-patients from about the fourth century downwards. Many of the public dispensaries were large buildings with a numerous staff of attendants.

In the Roman City and Empire, too, some time before our era, there were clearly great and well-equipped hospitals, *valetudinaria*, for well-to-do persons as well as for the poor and slaves. The staff attached to one of these hospitals included not only medical men but also assistants, dressers, and librarians or registrars. It is curious to find Celsus speaking of doctors at large hospitals having so many patients that they can hardly give them the individual care they need. On the building, cleansing, decoration, and cookery thought best in these *valetudinaria*, we have actual treatises by Columella, Vitruvius, and others. As to military hospitals, though there is plenty of evidence of a medical service in Greek armies and possibly in Persian ones, there is no mention of hospitals; but in Rome from the time of Augustus the military hospitals were excellent and well arranged. Thus Haberling and Allbutt speak warmly of the one at Carnuntum, 25 miles from Vienna, in the first century, and of Novesium on the Rhine under Tiberius and Claudius, built on the corridor plan with many small wards. Under Hadrian we read of others.

Thus, in the pagan world, hospitals were founded (1) at sacred shrines, or (2) by municipal care, or (3) by private practitioners, or (4) by military rulers.

There is reason to think that between 100 and 300 A.D., in the social troubles of the Roman world, municipal and possibly other hospitals fell largely into decay. Then Allbutt states definitely that Constantine in 335 A.D. decreed that all public hospitals should be closed and their staffs dismissed, but later on he allowed some to be reopened. This reminds us of our Henry VIII and the London hospitals. One would like to be clear whether it was not solely directed against pagan shrines, if such a decree was issued, but I confess that I can find no evidence for the statement.

CHRISTIAN HOSPITALS.

In the Roman world great changes and a remarkable expansion of the hospital system gradually took place under Christianity.

1. Municipal hospitals are little heard of for a very long period, but the official hospitals of the bishops become important.

2. Military ones under Byzantine rulers are said by Bury to have been good and well equipped; otherwise we hardly hear of them till the time of the Crusades.

3. The emperors and kings founded a fair number of civil hospitals.

4. The sacred shrines had successors in some Christian churches.

5. Though the *iatreia* of some private doctors long survived, medicine and the other arts died out almost entirely in large areas.

6. Individuals and voluntary subscribers founded vast numbers of hospitals wherever the barbarian invaders and the wars allowed.

7. After 529 A.D. monasteries took up the work in Italy, and later on in the northern countries.

The Bishops' Hospitals.—In the first age of Christianity the house of every Christian might be used as an infirmary, as Pelliccia says,⁷ but from the time of Constantine or earlier it was customary for each bishop to set up a hospice with beds for the sick close to his own house and cathedral.⁸ This is repeatedly mentioned. Thus the fourth council of Carthage, 436, made it compulsory for that region, the hospice to be *non longe ab ecclesia*. Sometimes the bishop managed it personally: thus we find Augustine taking his meals among the sick; usually he appointed one of his canons to do so. Later on in Rome a regionary deacon, and still later a monk, was put in charge. The council of Chalcedon insisted that the manager of a hospital must be under the control of the local bishop. Like episcopal schools they were supposed to exist in every diocese, but their success was variable. In some places the bishop was too poor to provide more than a rest-house; in others a good hospital appeared, and even a school of medicine.

Charlemagne in his capitularies of 803, and the Councils of Aix la Chapelle 817 and 836, lay down that a hospital must be attached to each cathedral and even to each collegiate church and monastery. At Orleans in 549 it was enacted that the bishop must maintain the hospital which Childbert had founded. At Lyons in 583 the bishop of each see is required to provide for and segregate the lepers. Notable instances of episcopal hospitals are seen at Cæsarea under Basil in 370, Merida in Spain 580, where the great revenues and the physicians and nurses are mentioned, as well as the fact that Christians or Jews, slaves or freemen, were all taken in and treated. Then we have Chrysostom's foundations at Constantinople, one at Ephesus, another at Hippo, and in a later age the Hôtel Dieu at Paris long managed by the Chapter of Notre Dame, those at Chalons, Tours, Rheims, Cologne, Bremen, Constance; and in England at Worcester, Lincoln, Canterbury, and Winchester. As we shall see later on, the management was finally handed over to the lay magistrates and revived municipalities in many countries.

Under Justinian startling legislation took place as to the bishops in other matters. Roman law had previously provided very little protection for charities against fraud and peculation. Justinian now took the bishops and made them guardians and public trustees of all charities, hospitals included. Thus, if a legacy appeared in a will to found a charity, it became the bishop's duty to appoint an officer to enforce the proper performance of the legacy, and they received power to visit all institutions and to correct abuses.⁹ All this is quite distinct from their duty to found and manage their own diocesan hospital. In the breakdown of the State this visitorial power became the only protection charities had, but it was a great tax on the time of the bishops and their councils. We find it lasting all through the Middle Ages and asserted in the Councils of Vienne, 1311, and Trent, 1545. Thus the Council of Durham, 1217, said, "Those who wish to found a hospital must receive from us rules and regulations", much as they would apply to-day to the visiting justices.

Imperial, Royal, and Papal Foundations.—Of these may be noted especially those at Rome by Symmachus in 500, by Belesarius in 550, and by Pelagius II in 560, at Lyons by Childebert about 500, at Autun by Brunelhaut in 595, at Jerusalem by Justinian about 510, and again at Constantinople, where he gave up one of his palaces to form a new one. That town obtained another from Isaac II in 1190. Alexius had already in 1116 rebuilt and enlarged a huge infirmary there, said to shelter 10,000 inmates. Our king Athelstan had endowed or founded St. Leonard's at York in 937. This had afterwards 220 beds as well as an orphanage under the same roof.

Christian Shrines.—After the coming of Christianity the populace long adhered to the Asclepian shrine treatment and worship. To meet the difficulty various churches were built, and the people were induced to resort there to implore the intercession of the saint or martyr commemorated in them, the theory being that the One God the Healer and Saviour of all men might grant a cure in response to the prayer of his servant the saint or martyr. Crowds of sick came to pray, and many claimed to be cured. Even incubation in its old form soon appeared, and the kindly saint was believed to give advice in visions to those who begged for his intercessions with the Almighty. Here and there followed hospices for the waiting sick in which baths and medical treatment were given. Thus the churches named after Cosmas and Damian replaced the shrines of Castor and Pollux, Cyrus and John drew off the worshippers of Isis or Manuthes, and the prayers of St. Michael were sought at Sosthinium in place of the cures of Apollo or Serapis. This cult has survived to the present day, with hospices at Cyzicus, Daulia in Parnassos, and with incubation at Tenos, to mention only a few.⁵

Monastic Foundations.—We need not spend much time over the well-known and great work done by the early orders. St. Benedict gave a very high place to the care of the sick. *Infirmorum cura ante omnia et super omnia adhibenda sit*, says his rule. Cassiodorus urged the need of medical study and collected a little library of medical books for the brethren. The infirmaries for the monks, and in some places for outsiders, were built with extraordinary care, and regardless of expense; they began in Italy from 529 and spread everywhere there, but not in France, Germany, or England until about 800, and during the utter breakdown of civilization the monks almost monopolized medicine. It has been said that Benedictine houses reached 37,000 in number. Whether that is true or not, in the Cluniac branch every house was ordered to build an infirmary for monks and a hospital for outside patients. Of course there was always a minority, like the Christian Scientists and others to-day, who were opposed to medical treatment. These relied on prayers and charms, but that does not affect the enormous sums which the majority spent on hospitals for the sick, and such medicines as they had. In England, the monks of St. Alban's built their infirmary in 794, and it was followed by a host of others. In many places, where no other doctors existed, there was a constant drain on a convent to send out skilled monks to attend the sick.

Individual Founders.—Besides the official work of the Christian society, it was the first duty and privilege of each individual to care for the sick and poor. Compassion for the sufferers, says W. H. S. Jones, was a virtue amongst Pagans: it was *kalon*; but amongst Christians it was *deon*, a duty. Hence

occurred a huge new outburst of hospitals founded by individuals or by voluntary subscribers as soon as it could be safely done. For example, Zodiëus, about 330, collected the lepers in Constantinople into a hospital, the wealthy Olympia and others helping in the work. Hospitals there soon multiplied so fast that Ducange has reckoned 34 hospitals and asylums in it, but many of these were gifts from kings and bishops. At Edessa, in 372, St. Ephrem collected subscribers for a hospital of 300 beds, which was refounded in 410 by Bishop Rabbouli with another for women. Here a brilliant medical school grew up which carried Greek medicine to the Far East. At Rome, Fabiola in 380 gave her wealth to a new hospital in which she worked as a nurse. St. Paula did the same near Jerusalem. We find St. John, the almsgiver, building seven hospitals for women at Alexandria.

To take another instance from the charming history of Sorore of Sienna about 898, the William Law of his time. How he began with one poor wretch lying in the street, and gradually built the great hospital of St. Maria del Scala, and got together subscribers, a staff, and a lay committee, all so well organized that ten other Italian cities came to get him to introduce his system and trained helpers there also.¹⁰ Lastly, as in private duty bound, I pray you to remember our own Rahere founding St. Bartholomew's in 1123 with help from rich and poor alike. These are only a few instances of the munificence of the war-stricken and dismembered Christian world.

MOSLEM HOSPITALS.

These mark an important stage in our story, and since the empire of the early Caliphs soon became highly civilized and wealthy, hospitals and the medical art made extraordinary progress. The relief of the sick and poor was also a duty in Islam, and the new state drew its inspiration as to medicine and hospitals from two sources. First, there were its Byzantine neighbours, who had countless hospitals. Then, besides this, Nestorian missionaries, doctors, and entire medical schools had been exiled to Asia. It has been said that Syrians and Egyptians, being strong home-rulers, took up any heresy condemned by Constantinople, and when persecuted went over the border with their Greek literature and science, or in later times often supported the Moslem invaders. The Persian kings gave some toleration to the earlier fugitives, and their medical skill won them favour. One group settled at Gondishapor and joined the staff of the great school and hospital there. As we have seen, Chosroes by his embassy to India obtained for it the teaching and remedies of the then flourishing Buddhist hospitals. This school, derived from Greek, Persian, and Indian origins, was the second source of Moslem knowledge. Even the Pagans of Harran were propagators of Greek medicine and translators of texts. Al Harith, one of the alumni of Gondishapor, attended Mahomet himself.

The first hospital of the Caliphs was one set up at Damaseus in 707 A.D., by Al Walid, for eye cases, for lepers, and for the ordinary sick. His cousin Khalid had studied chemistry under an Alexandrian teacher, and we may ascribe this hospital to Byzantine influence. The invaders of Spain quickly founded a hospital at Cordova, and in Egypt, in 873, Tulun built one at Cairo,

and used to go round the wards himself to inspect them, till one day a mentally deranged patient threw a ripe pomegranate at him and spoilt his robes.

Turning back to the other source, we find the Caliph Al Mansur, in 765, sending for George Bukht Yishu, the chief physician at Gondishapur, to attend him.¹¹ Other physicians and also translators of Greek medicine followed, and hospitals sprang up so fast that about 790 Haroun Ar Raschid actually ordered that a new hospital should be erected for each fresh mosque in Baghdad, curiously echoing the words of his distant friend Charlemagne. Again, in 840, we find the mother of the then Caliph is founding another, and in Persia, now a Moslem country, a great hospital appears at Rhay or Rhazes near Teheran. Here we learn that Abu Bakr, afterwards so famous in Europe as Rhazes the physician, was chief of the staff with pupils and clinical clerks. He, too, was sent for to Baghdad to preside over the great Jahya hospital about 905. Two fresh ones were also erected, and the Jahya itself was rebuilt with great magnificence by the Caliph Azudu'd Daula in 976, and then was known as the New Hospital near the bridge on the west of the city.¹² It had a staff of 24 physicians, and special wards for fevers, eye cases, accidents, and various other diseases. The Caliph also determined to suppress quacks, and gave a commission to his minister Sinan to examine all practitioners, and license such only as he thought fit. As no less than 860 candidates presented themselves, Sinan's task was no sinecure, and we learn how popular medicine must have been at the time.

Another great physician of this Azudi or Jahya hospital was Ali ibn l'Abbas. He writes forcibly of the importance of students attending hospitals regularly with the professors, and noting the condition of the patients and all their symptoms, while at the same time they bear in mind the classical symptoms of disease as given in their text-books, and compare the two pictures. Indeed, medical studies were carried to a great height at this time. Not only did the leaders possess a good knowledge of Greek, Roman, and Indian writers, but by endless observations and case histories they built up a real advance in our knowledge.

In Moslem Spain new hospitals were added to the old ones at Toledo, Seville, and Algeciras, as well as at Cordova, where we find the Sultan Hachem regulating and reforming them, and appointing an examining board to license state physicians and the hospital staffs. Indeed, hospitals began to appear everywhere, from Merv and Mosul in Asia, Mecca and Medina in Arabia, Harran and Aleppo in Syria, to distant Fez in Algeria, Shiraz, Hamadan, and Ispahan in Persia—not to forget Saladin's foundation in Cairo.

Three of the greatest Moslem hospitals were at Tabriz, Damascus, and Cairo. The first of these was built by the great Vizier Rasidu in 1295. He sent agents to foreign countries to collect good or rare drugs, and built a huge model hospital, with a staff of surgeons, oculists, and bone surgeons, each served by five dressers; and thirty physicians, each having ten students as clerks. However, this had a short life, for his enemies in the troubles of the time succeeded in ruining him and destroying his beautiful hospital.

The Bimaristan at Damascus was built by the fierce warrior Nur ud Din after his victories over the Byzantines about 1160, and its comforts and splendours were open to rich and poor alike. This was more lasting, and for 300

years it is said that its fires were never extinguished. Here, too, was a great medical school, for which another block of new buildings was added in 1250.

It was when lying sick in this palatial hospital that a vow was made by the Sultan of Egypt, Kalaoun, that if he recovered he would build another in Cairo. The result was the noble Bimaristan which he opened in 1284. One still sees to-day its small but exquisite mosque and tomb chapel in perfect order. It had three courts with trees and splashing fountains, the chief court had a verandah into which opened the large wards, while the smaller courts were surrounded with little private ones. There were, indeed, wards for every disease known, laboratories, baths, lecture rooms, a dispensary, and a library with its five assistants. There were convalescent homes or allowances for those recovering, and an income of £25,000 per annum. At the present time part of this ancient house has been made into a very up-to-date eye hospital with male and female wards, operating theatre, X-ray and other installations, under the able care of Dr. Mohammed Khalil, late chief medical officer of the ophthalmic hospitals. Another fine Cairo hospital, El Moayad, now being restored by the State, did not arise till rather later, and must be omitted here.

It is difficult to classify these Moslem hospitals as to their founders. Most of them were due to military rulers, but individuals created many, and possibly some are due to bodies like Dervishes. Shrines, though existing everywhere and thronged by crowds of sick, have not, so far as I know, grown into hospitals.

To return to the European hospitals, in which remarkable changes began about 1100. We have seen that, besides endless foundations by individuals and some by princes, great numbers had been built and managed by bishops, canons, and monastic orders, and that, in the scarcity of lay physicians, canons and monks had largely monopolized medicine. The church found the drain too great, and that large numbers of her official staff were neglecting their proper functions, often for the sake of making money, to study and practise as secular lawyers, physicians, and pharmacists. It had to be stopped. Decrees by some ten councils and popes, 1181 to 1234, forbade the abuse, and the ecclesiastical practice of medicine was greatly reduced.¹³ Fifty years later these stringent laws were relaxed under safeguards, but the check had been given. It is a curious thing that historians have often taken these councils as forbidding surgery to priests, which is never mentioned in the seven earlier ones, such as Tours. Only in 1215 does the prohibition creep in, to be relaxed with the others later on.

To fill the gap, besides the many lay doctors whom the fresh Universities and Schools soon provided, some 20 new societies of laymen to found and work hospitals were sanctioned. An enormous demand arose. It was the time of the epidemic of leprosy (1100 to 1350), the time of the Crusades, the date of a great religious revival, and of the revival of Guilds which were keen to take up any charitable work. Hospitals became the passion of the age. Individuals founded them everywhere or subscribed to an order of Hospitallers to do so.

Of these orders or hospital societies, three or four combined fighting with their work, and thus revived the military hospitals as well as civil ones.

1. *The Teutonic Knights*, besides their Palestine houses, founded sixty hospitals in towns in the Baltic area, such as Dantzic, Thorn, Elbing, and Königsberg.

2. *The Templars* founded others; for instance, those at Frankfort-on-Oder and Munchaberg.

3. *The Knights of St. John of Jerusalem* inherited an old hospital there, and about the year 1120 built a fine new one with 2000 beds under the care of five physicians and three surgeons, the former having to give proof of their skill in pharmacy and uroscopy. When the knights were driven out by Saladin they built other hospitals at Acre, Rhodes, Malta, and other places in Europe. Thus they took over and worked a large previously existing hospital at Salerno. Their house at Malta became perhaps the finest and best equipped hospital in Europe until it was plundered by Napoleon. Later writers tell of the patients in their snowy white cubicles, delicately fed, and served on silver plate.

4. *The Lazarists*, whose history is obscure, are said to have helped with leper hospitals, of which there were 200 in England and 1700 more on the Continent. I do not think that these were wholly quarantine stations. Treatment and even cures occurred, and it seems that these houses were sometimes used for other infectious diseases, so that the name of lazar house is then given to what we should call a fever hospital.

5. *The Order of St. Antony* met the outbreak of ergotism with houses in England, France, and Italy.

6. *The Society or Order of the Holy Spirit* was perhaps the most active of all.¹⁴ Founded about 1170 by a Mr. Guy at Montpellier, it was so well managed that Innocent III gave it great privileges in 1198. It was a purely lay body of men and women for founding hospitals and nursing "*Our Lords the Sick*". By 1300 it had raised 900 hospitals. One branch worked in Hungary, Italy, and England. The Pope built for them the still existing Hospital of the Holy Spirit in Sassia at Rome on a site given by the English king where King Ina of Wessex had founded a rest-house 500 years earlier. The revenues of this hospital reached in after times the sum of £37,500 a year, and it is even now a fine building. The other province included France with 400 hospitals, certain other countries, and Germany, where Professor Virchow traced 155 hospitals of the Holy Ghost, their headquarters being at Montpellier. In England their records have been entirely lost, and though King John gave them an estate at Writtle, in Essex, and the bishops licensed them to collect alms, no hospital has been traced to them with certainty, although several bore that dedication, and one of these was amalgamated in 1436 with St. Thomas's in London. Besides their whole-time members, the Order had an enormous number of subscribers who raised funds and visited the sick, forming the Confraternity of the Holy Ghost. This had branches in Bristol and elsewhere, and in after days King Henry VII, the Lady Margaret, and John Colet were among its members.

I have not time now to speak of other societies of hospitallers—Albrac, Roncesvalles, St. Gervase, etc.—or to catalogue the many municipalities, guilds, and individuals who founded hospitals on the Continent at this time,

especially in such towns as Florence and Cologne. We may just notice that in England between 1100 and 1300 four hundred new hospitals and asylums were founded either by individuals, guilds and societies, or bishops and kings. This is in addition to the monastic infirmaries. Some of them were hospitals in the strictest sense, such as St. Bartholomew's, and we must not forget St. Thomas's in London, and those at Gloucester, Lincoln, Norwich, and Durham, and the older ones at York and Canterbury.

Finally, over Europe generally, since lay efforts had shown themselves so efficient, and since municipalities in some places had taken up public health as in Greek times, the decrees of the Councils bore fruit, so that a movement arose to hand over the management to lay magistrates. This was done in many large towns and in Rome itself, by the joint wish of all parties, about 1300, though somewhat later in France.

We have, then, briefly traced the growth of hospitals up to this epoch, just before the Black Death. One is struck with the great number of men of various races, religions, and civilizations who have laboured at the work. Then, when one thinks of the vast number of hospitals which have perished, regret must arise that the life of hospital systems is so fragile and so easily destroyed by war and by changes in fashion, in government, in religion, or medicine, and that such a huge volume of work has been thrown away by their early decay.

Is not this just what we find to-day, that invaluable as hospitals are, they are no sooner built than they are superseded, or need alteration? In fact they are typically "a short term investment".

REFERENCES.

- ¹ SMITH, V. A., *Life of Asoka*, 62 and 165.
- ² WITHINGTON, *Medical History*; and WIGESINTHA, *Mahavansa*.
- ³ SMITH, V. A., *History of Early India*, 312.
- ⁴ DHALLA, *Zoroastrian Civilization*, 352.
- ⁵ HAMILTON, MARY, *Incubation for the Cure of Disease*.
- ⁶ ALLBUTT, SIR CLIFFORD, *Greek Medicine in Rome*, 460.
- ⁷ PELLICCIA, *Polity of the Christian Church*, 533.
- ⁸ EPIPHANIUS, *Adversus Hæres*, bk. liii; *Burdett's Hospitals*, i, 37.
- ⁹ *Codex Justiniani*, lib. I, Title ii, cap. 15, 19, 23, Novellæ 120, 131; BUCKLAND, W. W., *Text-book of Roman Law*, "Pæ Causæ", 178, 179, 256.
- ¹⁰ HELYOT, *Histoire des Ordres Monastiques*.
- ¹¹ BROWNE, E. G., *Arabian Medicine*, 45, 56, 109; O'LEARY, DE LACY, *Arabian Thought*.
- ¹² USAIBIA, "Life of Rhazes," in *Proc. International Congress Medicine*, 1913, sect. xxiii.
- ¹³ MANSI, *Tomes xx-xxiii*, Councils of Rheims, London, Lateran II, Montpellier I and II, Tours, Paris, Lateran III, also Letter of Honorius III, Decret. Gregory IX and Boniface VIII, Sext. Decret.
- ¹⁴ SAULNIER, P., *De Capite S. Ord. S. Spiritus*; and BRUNE, P., *Histoire de l'Ordre Hosp. de S. Esprit*; also Patent Rolls, Papal Letters, etc.

URETHRAL DIVERTICULA.

BY T. B. MOUAT,

ASSISTANT SURGEON TO THE ROYAL INFIRMARY, SHEFFIELD.

URETHRAL diverticula may be congenital or acquired, and the causes of the acquired variety vary in the different portions of the canal.

In the prostatic urethra, pouches of this kind are most commonly acquired from the gradual increase in size, and communication with the urethra, of a sac or sacs containing calculi which formed in the gland substance.

In a second and less common variety of acquired diverticulum, the prostatic urethra may gradually become distended to form a smooth-walled sac as a result of the lodgement of a stone or stones in that portion of the canal; two of the three cases recorded by Morton,¹ and the following case published by Ferraton,² appear to have been of this kind.

The patient, age 24, a quartermaster in the Navy, had been treated for pain and difficulty in passing water, at the age of 4 years. Fourteen years later these symptoms recurred, and troubled him at intervals for the six years preceding the operation in 1904. An ovoid phosphatic stone, with a core of urates (25×18 mm., weight 5.697 grm.) was removed by the suprapubic route through the bladder, from a smooth-walled sac formed by the prostatic urethra.

Ferraton thought that the cavity was preformed and probably congenital in origin, because of the long history, and the disparity in size between the sac and the stone; but it seems more probable that the pouch was acquired about a stone which lodged in the prostatic urethra in infancy.

As rarer causes of prostatic pouches, it has been suggested that cysts or abscesses of the gland substance might burst into, and persist as acquired diverticula communicating with, the canal.

CONGENITAL PROSTATIC DIVERTICULA.

The congenital prostatic diverticulum is less common, and while several of the older writers described and figured prostatic diverticula which might well have been of this nature, the suggestion that a pouch in this region might be the result of the gradual distention of the sinus pocularis is of comparatively recent origin, and in our own literature appears first in an excellent paper on "Urethral Calculi, with Special Reference to Encysted Calculi of the Prostatic Urethra", published by Monsarrat³ in 1912. While that author considered that the diverticula in his three cases were most probably acquired, from the gradual expansion and fusion of pockets which contained prostatic calculi, he discusses the possibility of congenital origin, and concludes that "it is impossible to negative the presence of a preformed diverticulum as the first stage of the whole process, in view of the records of such preformed pouches in this part of the canal".

Bourdillat's¹ thesis on *Calculs de l'Urèthre*, which appeared in 1869, contains the illustration of a prostatic pouch, here reproduced (Fig. 34), which might have arisen from distention of the sinus pocularis, for the position of the diverticulum, and the fact that it contains a single stone, are rather characteristic of the congenital variety.

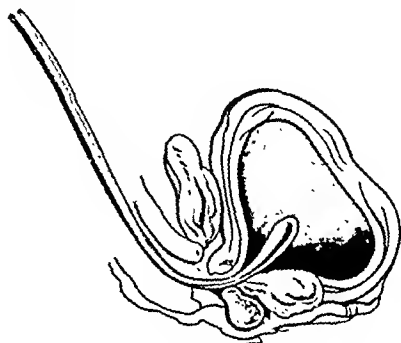


FIG. 34.—Stone in prostatic pouch.
(From Bourdillat, *loc. cit.*)

Bourdillat states that this illustration was borrowed from Crosse's *Treatise on the Urinary Calculus*,⁵ but it does not figure in the copy which is now in the Royal Society of Medicine.*

It appears to be probable that the sac had thus arisen from gradual distention of the sinus pocularis in the following case, which has recently been under my care :—

Case 1.—Two-ounce stone in sac which communicated with the posterior urethra.

S. F., age 48, complained of increasing frequency and difficulty in passing water.

HISTORY.—He had served in the Royal Field Artillery from 1900 to 1919; in 1902 he had gonorrhœa, and was in hospital for six weeks and discharged as cured. Two or three years later he began to be troubled by pains in the perineum which were brought on by riding. His present symptoms have been noticed for the last two or three years, and have gradually got worse. He passes urine every two hours during the day, and about five times each night. "The water comes sharp at first, then stops sudden, and then starts again"; and for some months he has been troubled by sharp pains above the left testis, which are brought on by coughing.

He has been married for ten years. No children, no abortions. There was no history of trauma; no history of difficulty or incontinence in his youth, and there was a complete absence of any previous history of renal colic. Nothing abnormal had been noticed as regards his sexual functions.

He was sent up to out-patients as a case of stricture of the urethra, but on rectal examination a very large hard mass could be felt in the perineum, between the posterior urethra and the rectum, which was pressing on and obstructing the urethra.

OPERATION.—He was admitted to hospital, and on Sept. 8, 1927, the hard mass was cut down on from the perineum, and proved to be a large phosphatic stone, 2 oz. 15 gr. in weight, occupying a large

smooth-walled pouch which lay between the posterior urethra and the rectum, and communicated with the urethra near the apex of the prostate, by a median slit

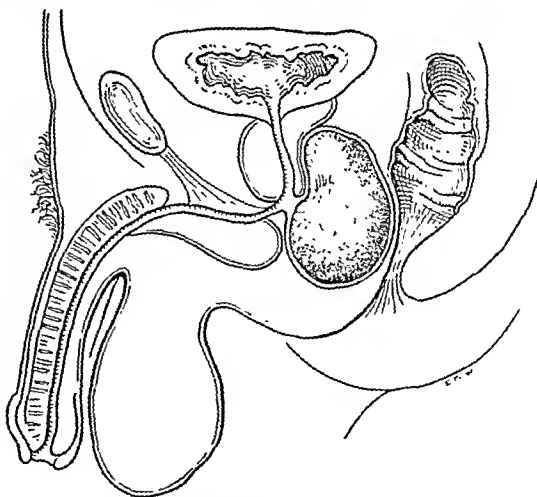


FIG. 35.—Case 1. Diagram of posterior urethral diverticulum and stone.

* John Green Crosse was Surgeon to the Norfolk and Norwich Hospital, and published his work entitled *A Treatise on the Urinary Calculus* in 1835.

about half an inch in length (*Fig. 35*). The stone was deeply grooved on the surface which corresponded to the gap in the floor of the urethra, and formed a beautiful mould or cast of all the structures in contact with it (*Figs. 36, 37*).

The pouch was excised, and the edges of the aperture of communication with the urethra were stitched together over a gum-elastic catheter inserted into the bladder. The progress after operation was quite satisfactory. The perineal wound leaked urine for some days after the catheter was removed, but later healed, and at the present time a 10-13 bougie can be passed along the urethra into the bladder without any difficulty.

I am indebted to Dr. Guest, Pathologist to the Royal Infirmary, Sheffield, for the following histological report :

Sections cut through the wall of the diverticulum showed it to be composed of the following structures :

A lining membrane composed of definite stratified epithelium with well-marked prickle cells. This membrane is composed of two parts : a superficial, in which the



FIG. 36.—Anterior aspect, with deep urethral groove.

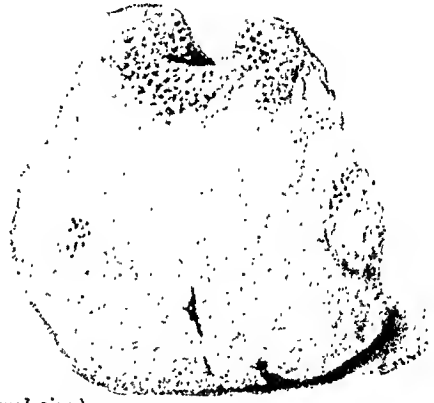


FIG. 37.—Posterior aspect, moulded by pressure of rectum.

cells are of polygonal shape ; and a deep part, where the shape of the cells becomes more elongated or cylindrical (*Fig. 38*). The most superficial cells are much flattened and their nuclei lost, and a tendency to keratinization can be seen in sections especially stained to show keratin. The nuclei of the deep cell layers took up hæmatoxylin much more readily than the superficial. Papillæ are well marked, and project upwards into the epithelium, and are composed of loose connective tissue.

Deep to the mucosa lies much fibrous tissue with scattered bundles of smooth muscle, the latter being more confined to the deep layers of the wall of the diverticulum, while blood-vessels are present in considerable numbers.

All layers show a round-celled infiltration, but this is more especially marked in the region of the subepithelial connective tissue. The vessels show some elastic tissue, and, throughout the fibrous and muscular tissue, fibrils of elastic tissue can be seen in suitably stained sections.

The stone which occupied the diverticulum has not been analysed, but on section it appears to be largely composed of phosphates, with some layers of urates, deposited around one central nucleus.

The grounds on which we base our hypothesis that the diverticulum was preformed, and had probably arisen from the sinus pocularis, which had persisted as a congenital defect that determined stone formation, and then enlarged *pari passu* with it, are as follows : the long duration of the history, and the complete absence of antecedent renal colic ; the position, anatomical

relations, and histological characters of the diverticulum, and the fact that the stone was single, and had formed around one central nucleus. The

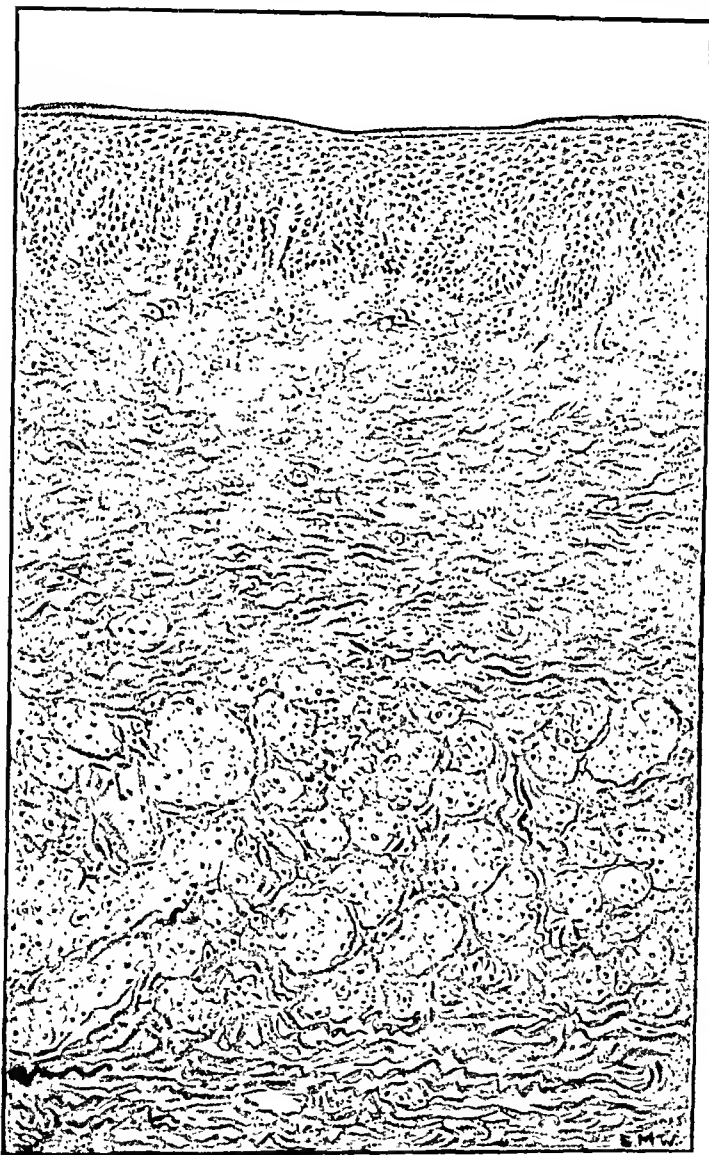


FIG. 38.—Case 1. Pen drawing of section of wall of diverticulum.

following somewhat similar cases, which have been recorded as examples of congenital prostatic diverticula, are cited in support of this opinion:—

Kapsammer⁶ found in a man, age 30, who had experienced difficulty in passing urine from his youth upwards, a diverticulum of the prostatic part of the urethra, “exactly like a vaginal sac”, in which a phosphatic stone, which weighed 162 grm., had developed. On microscopic examination the structure of the wall of the sac was found to resemble exactly a section of the wall of the vagina.

Escat,⁷ in an article entitled "Malformations Congénitales et Acquisées de l'Urèthre," describes and figures (Fig. 39) a most interesting and instructive case of which he gives the following particulars: A young man, age 18, had noticed, from infancy, a swelling the size of a nut situated in the mid-line at the junction of the posterior aspect of the scrotum with the perineum. This swelling always increased in size when he passed urine, and he used to press on it to empty it by the urethra. A few days before he presented himself he found that he could no longer empty the swelling, which became bigger and painful. The diagnosis of an inflamed congenital urethral pouch was made, and, after the pouch had been incised and emptied of pus and urine, an attempt was made to dissect out the wall of the sac, which narrowed down to a pear-shaped neck, from which a hollow stalk ran along the right side of the bulb, through the triangular ligaments, to end in the pars prostatica. A section of the stalk of the sac showed that it was lined by mucous membrane, enclosed in a sheath of unstripped muscle fibres. The mucosa comprised an epithelium and a dermis. The epithelium was stratified and resembled skin, with polygonal and cylindrical cells in the deeper layers; while, however, in the stalk the superficial layers showed no corneous change, and few and ill-formed papillæ, in the walls of the dilated sac the superficial cells showed keratinization, and there were well-marked papillæ in the dermis. There were no glands. The dermis was composed of adult connective tissue, with, in places, aggregations of lymphoid cells such as one finds in the walls of certain ranulæ and congenital branchial fistulæ. Outside the dermis there was a layer of unstripped muscle, and the stalk was enclosed by a fibrous sheath, which was firmly adherent to the layers of the triangular ligament.

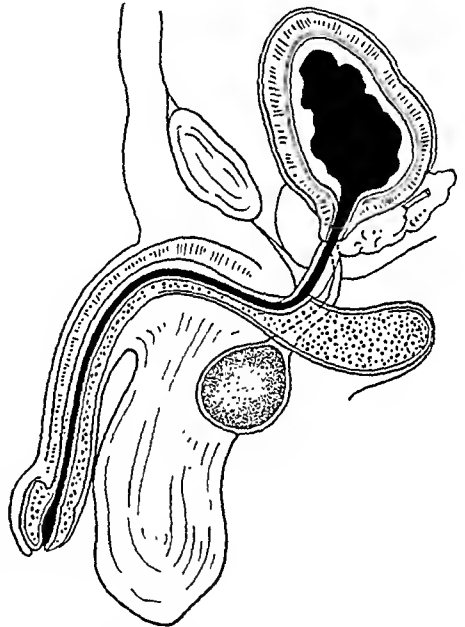


FIG. 39.—Congenital urinary pouch which communicated with the posterior urethra. (From Escat, *loc. cit.*)

Escat remarks that the above is an unusual example of congenital malformation, as the urinary pouch was developed slowly at the inferior extremity of a canal nearly 10 cm. in length. The hollow stalk was about the size of the round ligament, and measured 2 mm. in diameter, while the urinary pouch was about the size of a nut, and situated in the base of the scrotum. The author was unable to find a record of any similar congenital defect, but suggests that his case might be analogous to certain very rare congenital perineal fistulæ, two examples of which are referred to by Legueu in Le Dentu and Delbet's *Treatise on Surgery*; in both there was a tiny median fistula, which allowed a few drops of urine to escape at each act of micturition, and the fistulous tract, which was lined by epithelium, and enclosed in unstripped muscle, communicated only with the posterior urethra. Escat concluded that his case was a variation of the same malformation, and that the anatomical relations and histological structure suggested that it was a persistent portion of the Müllerian duct, which forms the vagina in the female.

Another probable example of a congenital urethral pouch, somewhat similar to Escat's case, was described by Thistle⁸ :—

A man, age 32, who had been treated for some years for a stricture of the urethra, was admitted to the Torbay Hospital, complaining of pain and swelling in the perineum, and of inability to pass urine. The perineal phlegmon was incised, and the central fluctuating swelling proved to be a very large inflamed urinary pouch, which extended into the perineum, and backwards behind the base of the bladder, and contained three uric acid stones which weighed 91, 124, and 425 gr.; the largest stone filled the back of the pouch behind the neck of the bladder, and had to be extracted with lithotomy forceps. The walls of the perineal pouch were rough and sloughy, and Thistle thought that it "was most probably formed by the dilated urethra behind the stricture and the walls of an abscess". The stricture was in the bulbous portion of the urethra, and admitted a catheter some weeks later, "when the parts had been kept at rest and all element of spasm has disappeared". The perineal wounds healed, but the patient was left with a perineal urinary fistula.

In addition to the cases quoted above, Finsterer⁹ and Schapiro¹⁰ have described examples of diverticula which communicated with the pars membranacea, and Monsarrat points out that the term 'membranous urethra' has not in fact been employed in so strict a sense by surgeons as by anatomists, and it is difficult to avoid the conclusion that some of these so-called membranous urethra diverticula are in reality diverticula of the prostatic urethra. A further difficulty appears to lie in the fact that the anatomical relations are disturbed by the growing pouch; the prostatic substance atrophies or is pushed aside, so that it may be a matter of real difficulty to determine into which portion of the posterior urethra the neck of the sac opens, and this difficulty must be still greater where the sac only communicates with the posterior urethra by a tubular stalk as in Esca't's case.

DIVERTICULA IN THE ANTERIOR URETHRA.

In the anterior urethra secondary or acquired diverticula are again much more common than the congenital variety; but where the complete absence of any previous history of colic appears to exclude the possibility that the stone descended the urinary tract to become lodged in the urethra, and when there is no stricture or other local lesion likely to give rise to urethral or para-urethral stone formation, the possibility that a pouch of this kind may have originated from a congenital defect in the formation of the urethra must be considered, and appears to be probable in the following case:—

Case 2.—Stone in pouch which communicated with the floor of the urethra at the peno-scrotal junction.

H. A., age 48, complained of gradually increasing difficulty in passing water. There is no frequency, but the stream has been gradually getting smaller in size, and for some months he has had to strain to pass it.

HISTORY.—The patient first noticed a hard lump under the base of the penis in 1917, but did not trouble about it till his present symptoms developed. He is married, and has three children, the youngest four years of age. There is no previous history of trauma, or of renal colic. He has never had retention, incontinence, or urinary symptoms of any kind before the gradual onset of his present difficulty. He denies ever having had gonorrhœa.

ON EXAMINATION.—There was a hard rounded swelling about the size of a walnut in the mid-line at the penoscrotal junction, which on passing a bougie was found to be caused by a stone lying in a pouch, which opened into the floor of the urethra.

OPERATION.—The patient refused to come into hospital, so on Sept. 9, 1927, the stone was cut down on, under local anæsthesia, and removed from the thin-walled

sac, which was tightly stretched over it. The small median aperture of communication of the pouch with the urethra was stitched up, and the thin-walled sac was obliterated by suture to form a pad, under the repaired floor of the urethra.

The position of the sac is shown in the accompanying diagram (*Fig. 40*). The stone weighs 158 gr.; the middle layers are composed of oxalates, while the outer and inner portions are phosphatic (*Fig. 41*).

In 40 cases of acquired diverticula of the urethra collected by Bourdillat, there was a history of some previous operation on the urethra (external urethrotomy, median or lateral lithotomy) in no less than 16, while in the remainder, false passages, blows or falls on the perineum, peri-urethral abscesses, and lastly all solutions of continuity of the canal such as 'crévasses spontanées' and 'poches urineuses' are mentioned as possible causes of the condition. Bourdillat, unfortunately, does not elaborate his suggestion that a urinary pocket might be the primary and determining cause for stone formation outside the urethra, and it was left to later writers to suggest that urethral diverticula might arise as a congenital malformation from defective fusion of the inner genital folds which form the floor of the anterior urethra.

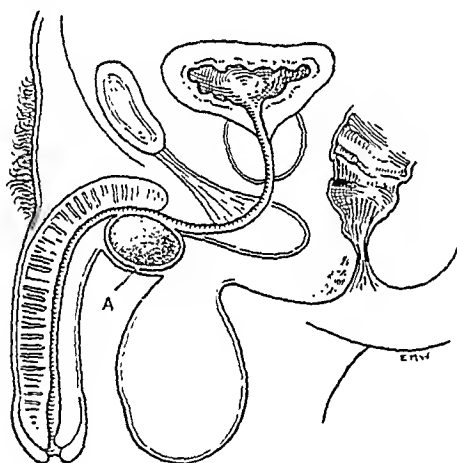


FIG. 40.—Case 2. Diagram of stone in urethral diverticulum (A).



FIG. 41.—Case 2. Stone. (Natural size.)

Eseat describes a congenital pouch of this kind, which opened into the floor of the penile urethra in a child of 5 years, and points out in discussing this case that all varieties of the defect in the urethral floor may be met with, from the gross malformation of hypospadias to minor defects such as blind internal or complete fistulæ or dermoids of the median raphe.

Erichsen¹¹ writes on the subject of urethral calculi: "In other instances they appear to be formed in a pouch that lies to the outside of the urethra, and that is only connected with it by a small aperture . . . one of the most remarkable instances of this kind is represented in the annexed cut (*Fig. 42*), taken from a drawing in Sir R. Carswell's collection at University College. The stone here was of very large size—equal in bulk to two horse chestnuts".

Hurry Fenwick¹² describes a similar case:—

A large stone, which measured $3\frac{1}{2}$ in. long, and 1 in. diameter, was removed by perineal section from a pouch in the floor of the penile and spongy urethra

in a man of 67 who had had difficulty in passing urine for eight or nine years. The anterior end of the stone was $2\frac{1}{2}$ in. from the external meatus, and only the nose of the stone projected into the canal.

In recent years Boominghaus¹³ recorded the following case:—

A man, age 36, who had suffered from retention at the age of 9, and a year or two later noticed a hard pea-like swelling on the under surface of the penis, which had recently become painful and swollen, and discharged urine and pus from

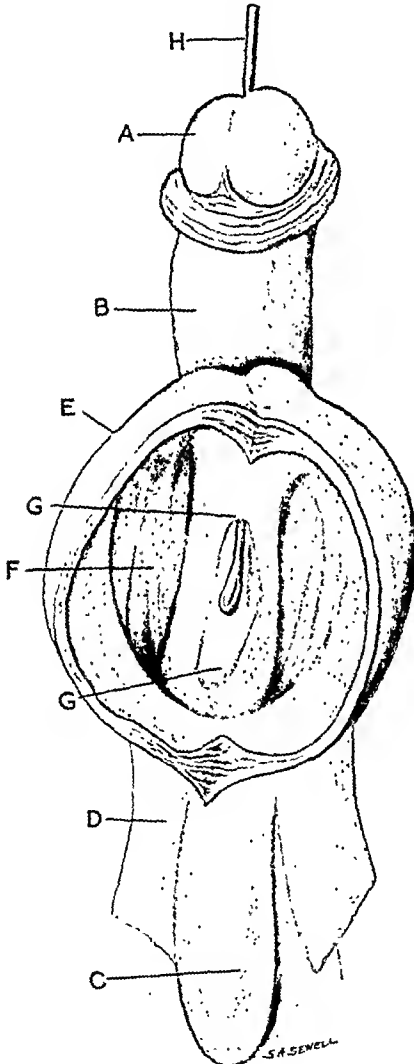
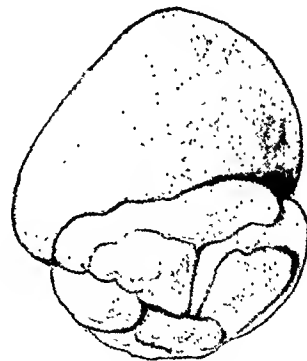


FIG. 42.—Dilatation of urethra in a case in Sir Robert Carswell's Collection. A, Glans penis; B, Body of penis; C, Bulb of the urethra; D, Crus penis; E, Wall of the dilatation, formed superiorly by the corpus spongiosum, laterally and inferiorly by the skin and cellular tissue; F, Cavity of the dilatation; G, G, The extent of the urethra communicating with the cavity. The probe (H) is introduced to render the direction of the urethra more apparent.

This cavity contained the calculus, and although the sound had been frequently introduced its presence had never been detected, probably because of the situation of the dilatation, which occupied the inferior surface of the penis, and because the stone did not project into the urethra. The patient was treated for sarcocele in the Hôtel Dieu, Paris.

The stone is shown separately.



fistulous openings. The swelling was incised, and a phosphatic stone, which measured $6 \times 5 \times 4$ cm., and weighed 136 gm., was removed from a large congenital sac lined by stratified epithelium, with well-marked papillae in the dermis, outside which was a layer of unstriped muscle enclosed by a fibrous tunic. The upper surface of the stone was deeply grooved by the urethra. The diverticulum opened into the floor of the urethra at the penoscrotal junction, by a median slit-like aperture 2 to 3 cm. in length. The sac was excised, and the edges of the opening into the urethra were sutured.

The description of a case which closely resembled the above was published by Nengebauer¹⁴ in 1924 :—

The patient, a miner by occupation, was 20 years of age. A small swelling, first noticed at the base of the scrotum when he was a year old, gradually increased in size, and later small fistulae formed in the right side of the scrotum, and discharged urine during micturition, at which time two swellings as big as plums appeared at each side of the penoscrotal junction. After the flow of urine ceased, he still felt a desire to pass water, which was relieved when he squeezed these swellings, and thus emptied them by the urethra and the scrotal fistulae. Fourteen small phosphate stones were removed from a diverticulum, about the size of a hen's egg, which opened into the urethra by a median slit-like aperture 1.5 cm. in length; while the lower end of the sac communicated through a narrow opening with a separate compartment, situated in the base of the scrotum, which contained another phosphate stone as big as a plum. Both the sacs were lined by stratified epithelium.

COMPOSITION OF URETHRAL AND PARA-URETHRAL STONES.

This has given rise to considerable discussion, for, while it is agreed that stones formed in the prostatic substance are composed of calcium phosphate, calcium carbonate, and organic material, and that any of the ordinary kinds of calculi may descend the urinary tract, to lodge secondarily in the urethra, there is by no means a similar unanimity of opinion as regards the composition of stones formed primarily in urethral diverticula, as the view expressed by several writers, backed by the authority of English¹⁵ that stones formed in diverticula must necessarily be composed wholly of phosphates, is opposed by other surgeons.

It is interesting to note that in 1835 Crosse pointed out that calculi formed in the prostatic substance might subsequently acquire deposits of urinary salts, if the pouches which contained them became so enlarged as to communicate with the urethra; while Monsarrat concludes that "given a preformed diverticulum, and the urinary constitution which tends to the production of calculi, there does not appear to be any difficulty in assuming that calculi other than phosphate may be formed *in situ*".

Our two cases appear to support the latter view, as the large stone (*Case 1*) contains some layers of urates, while the middle layers of the stone from the pouch below the anterior urethra (*Case 2*) are composed of oxalates.

DIAGNOSIS.

The diagnosis of urethral diverticula embodies two main problems: (1) The determination of the presence of a diverticulum; and (2) Its nature; and while it is frequently possible to decide the first from the clinical symptoms, which vary with the position, size, and contents of the sac, the second question can more often be settled only after operation and histological examination of the wall.

The diagnosis of a pouch opening into the anterior urethra is often fairly obvious, as the stone or stones which are usually contained in the sac can generally be felt in the perineum, or where the sac is not fully occupied by calculi it may become distended during the act of micturition, so that the

patient may have to press on the swelling to empty it through the urethra, or from the fistulæ which result from the septic infections which so frequently supervene in and around these diverticula communicating with the anterior urethra. The congenital diverticulum in this region may form a swelling which dates from infancy, while the causes of the various acquired pouches are usually determined by the presence of a stricture, or by a history of colic, operation, local injury, or disease.

The diagnosis is more difficult where the diverticulum is connected with the posterior urethra, though the surgeon to-day has the advantage that it may be possible to fill up a urethral or prostatic pouch with lipiodol or sodium bromide so that it will show in a radiogram; and in all cases the calculi, which are almost invariably present, may be thus demonstrated, even when they are embedded in the prostatic substance. In the patient of whom the skia-gram is given (*Fig. 43*), the prostatic concretions had been thus observed to grow, till the stones in one lateral pouch came to communicate with the



FIG. 43.—Skia-gram of prostatic calculi.

urethra, and it became necessary to remove them from the atrophied prostatic shell by median perineal section, because the patient got retention, and the spasm and intolerance to instrumentation, which, as Monsarrat pointed out, is such a troublesome feature in this type of case.

According to Erichsen, calculi in the prostate give rise to "a sense of weight, pain, and irritation in the perineum, sometimes to retention of urine, and in fact to the ordinary symptoms of enlarged and irritated prostate"; while Crosse wrote as

follows on the same subject: "When small and thus embedded in the ducts, prostatic concretions cause none of the characteristic symptoms of urinary calculi and cannot be detected; it is only when they increase and rise so as to project at the orifices of the prostatic ducts, or escape as they may do into the urethra, that they can be detected by the sound; but when of large size, or when numerous and contained in one cyst, they can be detected by the finger pressing on the gland through the rectum. . . . The pouch enlarges in the direction where there is least resistance towards the lateral or posterior surface of the prostate gland; thus in extreme cases the concretions are felt per anum lying close to the rectum, and in a cavity no longer communicating with the urethra".

The curious crepitation which may be felt by the examining finger is very characteristic, and has been compared to 'beads in a bag'.

When the diverticulum has been gradually acquired around a stone lodged in the prostatic urethra, there may be an old history of colic or bladder trouble, and the stone can usually be felt by a sound, while in other forms of diverticula a stone can only be detected in this way when it grows large

enough to project into the canal, or when a small calculus passes from the pouch into the urethra, where it may become impacted. In more than one of the recorded cases of prostatic pouches containing stones, this complication occurred, and when the stone was cut down on from the perineum it was found to have disappeared in a puzzling manner, as it had slipped back into the prostatic diverticulum.

TREATMENT.

The treatment of urethral diverticula necessarily varies; but where it is possible an attempt should be made to dissect out the wall of the sac, after it has been opened and emptied of its contents, while the aperture of communication with the urethra is closed by suturing lateral flaps, which have been dissected up at the neck of the sac, over a gum-elastic catheter passed along the urethra and left tied into the bladder.

This ideal method of treatment is generally possible—in the absence of any acute infection—in all varieties of diverticula of the anterior urethra, and also in the congenital prostatic pouch; whereas the most that can be done in the acquired forms of prostatic diverticula is to cut down and evacuate their contents, by a median perineal section. The stones are liable to re-form after this method of treatment, and for that reason Morton had to repeat the operation on one of his patients on six separate occasions.

REFERENCES.

- ¹ MORTON, C. A., *Brit. Med. Jour.*, 1906, ii, 294.
- ² FERRATON, *Arch. d. Méd. et de Pharm. militaire*, 1907, xlix, No. 1.
- ³ MONSARRAT, K. W., *Brit. Med. Jour.*, 1912, i, 3.
- ⁴ BOURDILLAT, *Calculs de l'Urèthre*, Paris, 1869.
- ⁵ CROSSE, JOHN GREEN, *A Treatise on the Urinary Calculus*, 1835.
- ⁶ KAPSAMMER, *Centralb. f. de Krankh. d. Harn. u. Sex.*, 1900, 18.
- ⁷ ESCAT, *Ann. des Mal. des Org. gén.-urin.*, 1908, i.
- ⁸ THISTLE, *Lancet*, 1892, ii, 1330.
- ⁹ FINSTERER, *Deut. Zeits. f. Chir.*, 1906, lxxxi, 140.
- ¹⁰ SCHAPIRO, *Annalen der russischen Chir.*, 1899, Heft. 5 (abstr. in *Centralb. f. Chir.*, 1900, No. 10, 280).
- ¹¹ ERICHSEN, *Science and Art of Surgery*, 6th ed., 1872, ii, 674.
- ¹² FENWICK, HURRY, *Trans. Path. Soc. London*, xli, 188.
- ¹³ BOEMINGHAUS, H., *Zeits. f. Urol.*, 1923, xvii, 535.
- ¹⁴ NEUGEBAUER, F., *Beitr. z. klin. Chir.*, 1924, cxxxii, 719.
- ¹⁵ ENGLISCH, J., *Arch. f. klin. Chir.*, 1903-4, lxxii, 487.

DIVERTICULOSIS OF THE APPENDIX AND PSEUDOMYXOMA PERITONEI.

By A. J. GARDHAM,

FIRST ASSISTANT IN THE SURGICAL UNIT, UNIVERSITY COLLEGE HOSPITAL, LONDON;

C. C. CHOYCE, C.M.G., C.B.E.,

DIRECTOR OF THE SURGICAL UNIT, UNIVERSITY COLLEGE MEDICAL SCHOOL;

AND MARTIN RANDALL,

MEDICAL OFFICER TO THE NELSON HOSPITAL, MERTON, WIMBLEDON.

DIVERTICULOSIS OF THE APPENDIX.

A. J. GARDHAM.

THE condition of diverticulosis of the appendix has received very little attention in English medical publications, although there are numerous references to the subject in the literature of other countries. It is for this reason that it seems appropriate to place the following case on record.

The patient, a man age 34, was admitted to University College Hospital under Mr. E. K. Martin on Jan. 25, 1927. The following particulars are taken from the case notes.

HISTORY.—Eleven weeks before admission to the hospital the patient had a sudden attack of pain in the lower abdomen, accompanied by nausea. The pain lasted some hours only, and then passed off, leaving nothing

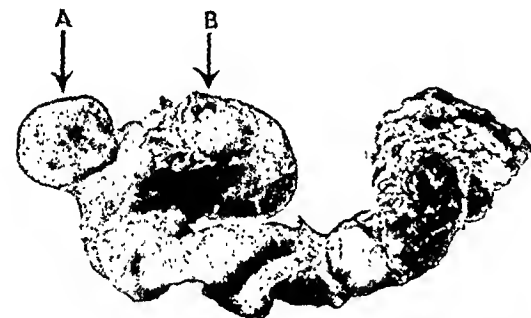


FIG. 44.—The appendix after separation of the omentum. A. Intact diverticulum; B. Ruptured diverticulum.

more than a feeling of soreness. Similar attacks recurred at intervals of two to four weeks. The patient was admitted to hospital in the fourth attack, eleven weeks after the first symptom. There was no history of abdominal pain previous to the present series of attacks.

PRESENT STATE.—The patient is a healthy-looking man. Tongue clean and moist. Temperature 98° – 98.8° ; pulse 80. Abdomen: There is a slight visible swelling in the right iliac fossa. On palpation a hard, slightly tender mass is felt, attached to the anterior abdominal wall. The mass does not extend to the limit of the peritoneal cavity laterally. Internally it appears to pass over the brim of the pelvis. Per rectum, a mass can be felt high up on the right side of the pelvis.

OPERATION.—Muscle-splitting incision over the right iliac fossa. The mass exposed consisted mainly of the great omentum. On separating the

great omentum the appendix was exposed lying to the outer side of the caecum. The appendix, which showed the condition described below, was removed, together with the omentum involved in the mass. No pus was encountered at any part of the operation. A drainage tube was left in the wound on account of the rather marked oozing of blood which followed the separation of the omental adhesions. The patient made an uneventful recovery and left the hospital on the fourteenth day.

After separation of the omentum the appendix showed the following condition (*Fig. 44*). In its proximal three-fourths the organ was of normal size. At the junction of the third and distal fourths was a spherical reddish projection the size of a pea, attached to the antimesenteric border. The distal fourth of the appendix was distended, and on its lateral side about one-third of an inch from the tip was a projection of mucous membrane, there being in this situation an opening from the surface into the lumen of the appendix. It is probable that this opening into the second diverticulum was the result of trauma in separating the appendix. The lumen of the appendix proximal to the diverticula was still patent, but so much diminished in size that an ordinary probe could not be passed, the patency being demonstrated by passing a fine bristle through it. The omentum removed with the appendix was very fibrous, but showed no mucoid material.

MICROSCOPIC SECTIONS.—

1. The unruptured diverticulum shows a clean-cut gap in the muscularis, through which the diverticulum protrudes. The diverticulum consists of a single layer of mucous membrane (as opposed to a number of cases quoted by Stout¹ in which the diverticulum consisted of a hernia of the complete mucous lining of the appendix). The covering of the diverticulum consists mainly of granulation tissue, a little fibrous tissue, and a few muscle fibres. The peritoneal coat has disappeared over the greater part of the diverticulum, and in these 'raw' areas numerous newly-formed vessels can be seen, as if the tissues of the diverticulum were engaged in picking up a new blood-supply from the surrounding omentum. The mucous membrane of the diverticulum is seen, on

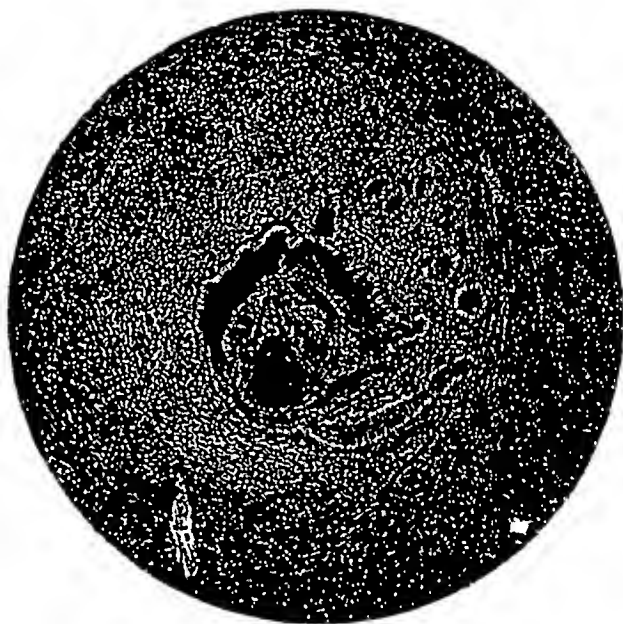


FIG. 45.—Section of the appendix proximal to the diverticulum, showing contracted lumen. ($\times 56$, $\frac{2}{3}$ obj.)

staining with mucicarmine, to be actively engaged in the production of mucus.

2. The surrounding omentum shows numerous strands of fibrous tissue. Mucicarmine reveals no mucus.

3. A transverse section of the appendix proximal to the diverticulum shows a definite but extremely contracted lumen. The diminution in the lumen is due to an increase in the fibrous tissue in the submucous coat (*Fig. 45*).

COMMENTS.

The points which I wish to emphasize in this record and which I believe to be characteristic are, firstly, the history of repeated attacks of very slight intensity, and secondly, the presence of a fixed mass in the appendix region in the absence of any of the ordinary signs of inflammation (compare with Neumann's² and Trotter's³ cases).

The main interest of diverticulosis of the appendix lies in the relation of the condition to pseudomyxoma of the peritoneum. Diverticulosis of the appendix appears to be a condition of only moderate rarity; in addition to the case here described there are three appendices showing diverticula in the Museum of University College Hospital. Moschcowitz⁴ found the condition in only 4 of 1500 appendices investigated, and McCarthy and McGrath⁴ in 17 of 5000; but investigations by Stout appear to show that diverticulosis is of considerably greater frequency than this. It can, I think, be said with certainty that diverticulosis of the appendix is a much less rare condition than pseudomyxoma peritonei. On the other hand, those cases of pseudomyxoma peritonei which have originated from the appendix have been shown in nearly all cases in which the appendix has been fully investigated to be associated with diverticula; according to Neumann, 4 of the 8 cases of pseudomyxoma peritonei investigated by him showed diverticula of the appendix. It appears, then, that diverticula of the appendix frequently, but not necessarily, lead to a pseudomyxoma of the peritoneum. It remains to decide the additional factor which must be present for a diverticulum to lead to pseudomyxoma peritonei.

Two recorded cases of early pseudomyxoma peritonei, those of Neumann and Trotter, bear a very close resemblance to the case here recorded. All three gave a history of recurring mild attacks, and all three presented physical signs of the same type, namely, a mass in the right iliac fossa resembling the ordinary appendix abscess, but unassociated with fever, rapid pulse, or constitutional disturbance. In the case here recorded this combination of marked physical signs with mild symptoms was noted, but was thought to be accounted for by marked encapsulation of an abscess. In Neumann's case, as in my own, the mass consisted of omentum, and the appendix presented an unruptured diverticulum. In Neumann's case the lumen of the appendix proximal to the diverticulum is not known to have been obliterated; in mine it was definitely patent, although this could only be conclusively demonstrated by a microscopic section.

Recorded cases of diverticula of the appendix nearly always give a history of repeated attacks, usually of slight or moderate intensity, but in many an attack of greater severity led to the removal of the appendix (Chase,⁴ Stout¹).

It appears that the life-history of a diverticulum of the appendix is as

follows: One or frequently more diverticula are formed as a result of destruction of small areas of the muscle coat of the appendix by interstitial abscesses in the course of an attack of appendicitis. This destruction of muscle is probably a frequent event, but in the majority of cases the mucous membrane is involved in the destructive process, and the result is the ordinary 'perforated appendix'. If the mucous membrane is not destroyed a diverticulum results. In the majority of cases repeated attacks of inflammation lead either to perforation of the diverticulum and abscess formation, or to operative removal of the appendix.

In a minority of cases the inflammation dies down sufficiently to allow perforation of the diverticulum to occur without leading to abscess formation. These are the cases which ultimately develop pseudomyxoma peritonei. The actual changes which immediately precede the formation of the pseudomyxoma are not known, but the following theory appears reasonable to me.

The presence in many of the recorded early cases of an omental mass in the region of the pseudomyxomatous appendix, and the fact that pseudomyxomatous nodules in the omentum have come to be regarded as a characteristic sign in the early stages of the disease, suggest together that the omentum is closely connected with the production of pseudomyxoma peritonei. In the case here recorded the mucous membrane, in part at least of the diverticulum, seems to obtain its blood-supply from the omentum. Supposing rupture of the diverticulum to take place, it is possible that some part of the mucous membrane having already established a blood-supply from the omentum would remain to lead a parasitic existence outside the diverticulum. The logical sequence of this would be the continued production of mucus after removal of the appendix, a condition which has been recorded but not explained by those dealing with the fully developed condition of pseudomyxoma peritonei. The frequent absence of an epithelial lining in the diverticula in cases of pseudomyxoma peritonei also suggests that the rôle of the diverticulum is not an active one in the later stages of the disease.

I am indebted to Mr. E. K. Martin for permission to publish the case, which came under my care when acting as deputy for him.

REFERENCES.

- ¹ STOUT, A. P., *Arch. of Surg.*, 1923, vi, 793.
- ² NEUMANN, *Berl. klin. Woch.*, 1909, M.1, S.15.
- ³ TROTTER, W., *Brit. Med. Jour.*, 1910, March 19.
- ⁴ Quoted by CHASE, *Canad. Med. Assoc. Jour.*, 1927, April 17, 416.

SOME SHORT NOTES ON PSEUDOMYXOMA PERITONEI.

C. C. CHOYCE.

THIS condition in association with perforated ovarian cysts was first described by Werth in 1884, and since then has been noted by many others, but it was not until 1901 that Fraenkel described the first case in connection with a perforated cystic appendix. This was found, unexpectedly, at a post-mortem examination in a man of 79 who had died of cerebral softening.

Emil Ries reviewed the literature in *Surgery, Gynecology, and Obstetrics*, in November, 1924, p. 569. His summary shows that although all the early cases were in women, and associated with ovarian cysts, quite a number of the later ones were found in both men and women, and in connection with cyst formation in the appendix, or sometimes with diverticulosis of such cystic appendices.

In 1910 Trotter (*see above*) found in the literature 9 cases of appendicular origin and added another of his own. He found that three varieties of associated appendicular abnormality were to be seen: (1) Simple obstruction resulting from fibrosis (or, in one case, from carcinoma of the appendix); (2) Diverticula of mucous membrane through the appendicular wall, and subsequent rupture; (3) Multiple cystic degeneration of the wall of the appendix. In this connection it is interesting that an appendix removed in the University College Hospital recently showed diverticulosis, the diverticulum containing a mucoid material. This case is briefly recorded and illustrated above by Mr. A. J. Gardham. It is possible that if this diverticulum had ruptured, the condition of pseudomyxoma peritonei would have been set up.

It is a curious fact that some of the cases have been found to be associated in the same patient with both ovarian cysts and cystic appendices; moreover in at least one case there was a mucoid collection in an umbilical cyst due to non-obliteration of the omphalomesenteric duct.

In some of the cases in which a cystic appendix was also found, no communication with the interior of the appendix was demonstrated, but in Bailey's case recorded in *Surgery, Gynecology, and Obstetrics*, 1916, p. 219, there were perforations of both the ovarian cyst and the appendix. In many of the cases in which the appendix only was at fault, there was an actual perforation; in some there was evidence that perforation and subsequent closure had occurred.

The jelly-like material is variously stated as mucin or pseudomucin, and gives the mucin reaction with thionin; it is of the same character whether it originates from the appendix, the ovary, or both.

It would be supposed that removal of the source or sources, and removal of as much of the pseudomyxomatous material as is possible, would cause cessation of its development, but this is by no means always so, for several patients have had to be operated upon more than once. The jelly-like material, therefore, must either be capable of reproducing itself, or of being reproduced by cells that have been dislocated from ovary or appendix and implanted elsewhere in the peritoneal cavity. Careful search has been made for such cells by several writers, who have, however, been disappointed. No one has yet demonstrated any very definite clump of such cells, though in some cases chains of cubical cells have been found (as in Trotter's case). For the present it seems fair to assume that epithelial cells of this type may be the cause of recurrence. If that be so, there seems to be some evidence that eventually they die out, for several patients are alive and well a considerable time after repeated operations.

As regards the condition itself, it is difficult to improve upon Werth's description in 1884 as translated by Ries. He speaks of the "peritoneal cavity being filled with gelatinous material in its whole extent. This material is partly free and partly in the form of thick semitransparent layers lying

on and firmly attached to the abdominal wall and intra-abdominal organs. The masses contain delicate connective-tissue membranes and fine vessels. At times the gelatinous substance is enclosed in a delicate connective-tissue membrane, and forms, especially on the intestine, polypoid pedunculated structures". These statements exactly describe the conditions found in Dr. Randall's case noted below.

In many cases the peritoneal endothelium is intact under the adherent masses; in others endothelial cells seem to have grown over the masses as well.

As regards treatment and prognosis, it would appear that the ovary and appendix should *both* be suspect, and that if not healthy both should be removed; the coexistence of a cystic appendix with an ovarian cyst has been sufficiently common to cause certain writers, without a great deal of justification, to speculate that either the ovary was secondary to the appendix or vice versa.

If all the gelatinous material can be removed without too much damage to the peritoneum upon which it is superimposed, this should be done; and in view of certain reported cases of pseudomyxoma arising from an umbilical cyst, it would be wise to examine the umbilical region also. No drainage of the peritoneal cavity should be done. Many of the early cases in which this was done died. Recurrence may take place, and second or even third operations may be necessary, but the general tendency nowadays appears to be towards survival of the patient if the case is seen sufficiently early for proper surgical treatment. In the case quoted above this stage had long been passed.

BIBLIOGRAPHY.

To the bibliography to be found in Emil Ries' paper (*see above*) another case by Wilfred Trotter (*see above*) should be added, and also the following since the appearance of Ries' paper.

- CUSCADEN, *Med. Jour. of Australia*, 1926, June 19, 704. (From appendix in female.)
- LEHMANN, K., *Hospitaltid.*, 1926, April 8, 326.
- SOUTHEY and WEBSTER, *Med. Jour. of Australia*, 1926, June 19, 704.
- KOERNER, J., *Centralbl. f. Gynäkol.*, 1926, Jan. 9, 83. (Origin of.)
- KLOTS, T. S., *Nederl. Tijds. v. Geneesk.*, 1925, May 9, 2112.
- MULLER, G., *Bratisl. Iekar. listy*, 1924, Jan., 152.

PSEUDOMYXOMATOUS CYST OF THE PERITONEUM FOLLOWING APPENDICITIS.

MARTIN RANDALL.

J. F. T., male, age 71, was admitted to Nelson Hospital, Merton, on May 25, 1927. The complaint was of general weakness and great abdominal enlargement, with intolerable discomfort and distress.

HISTORY.—About three years ago patient had an illness, with abdominal pain of fairly acute onset for which no cause could be found. He was not gravely ill, but since then his abdomen has gradually enlarged. During these three years he has been an invalid, spending his time in house or garden, and much of it in bed. His appetite has been fair, and bowels regular.

ON ADMISSION.—Patient is pale and listless. He walks slowly and with difficulty, but can stand upright. There is no obvious difficulty in breathing. No sign of disease is found in mouth, lungs, or heart, and the urine is normal. There is no cedema of legs. Pulse 76; respiration 22; temperature 97·6°.

The abdomen is enormously and uniformly enlarged. The skin is not shiny, nor is the tension very great. The enlargement is such as to cause a direct forward projection of about three inches from the costal margin—the lower ribs being widely opened out. The whole abdomen is dull, and no resonance can be detected, even in the flanks. A fluid thrill is everywhere perceptible, but it is a somewhat sluggish thrill. Nothing could be felt on rectal examination beyond a general fullness of the pelvic cavity. An exploratory puncture made in the middle line below the umbilicus with a large abdominal trocar, only succeeded in obtaining a little gelatinous fluid, too thick for any considerable quantity to run through the trocar.

No diagnosis had hitherto been made, and this brought us no nearer one. At this stage Mr. Wilfred Trotter was kind enough to see the patient. He made a diagnosis of a myxomatous cyst originating from the appendix, and advised an exploratory operation.

OPERATION.—This was undertaken by me, on May 31, 1927, with the help of Mr. C. M. Brophy. A right paramedian incision with centre opposite the umbilicus was used. The cyst wall and peritoneum could not be differentiated, and a huge cavity was opened, full of thick stringy mucoid material, which did not run out freely, but had to be squeezed out. There were about 40 pints of the material. No bowels or viscera of any kind could be felt in the cavity, which proved to be that of a huge cyst. The patient was very far from well, so it was judged impossible to remove the cyst and it was closed, drainage appearing to offer no reasonable ground for hope. The patient gradually sank, and died on June 5.

POST-MORTEM.—An abdominal examination revealed the presence of a huge cyst, with a thick tough wall, the inside of which was almost black in colour. It extended to every recess of the abdomen and right down between the bladder and the rectum. Its connections with the surrounding structures were very intimate, and much force had to be used to remove it: it would have been quite impossible to remove it at the operation without a fatal issue on the table. The point of origin appeared to be in the neighbourhood of the cæcum; the specimen was sent to Mr. C. C. Choyce for investigation.

Description of the Specimen.—The specimen is shown overleaf in *Fig. 46*. Allowing for the reversal of right and left due to the fact that it is viewed from behind, it will be seen that the cyst fills the whole space from the ascending to descending colon laterally, and from the transverse colon to near the brim of the pelvis below. The posterior wall of the cyst has been largely removed. The cæcum is free from the cyst wall, and from it runs the appendix, also free up to a point an inch from its tip; this distal portion of the appendix passes through the cyst wall, and the tip of the appendix can be seen free, but lying amongst a mass of inflammatory tissue inside the large cyst. It is perforated at its tip. The inner aspect of the cyst wall is brownish in colour and much corrugated; it is everywhere coated with a

DIVERTICULOSIS OF THE APPENDIX 69

yellow grit. The assumption is that the contents of the cyst represented the secretions of the appendix over the years since his first complaint of symptoms. The grit on the cyst wall is composed of cholesterol and the

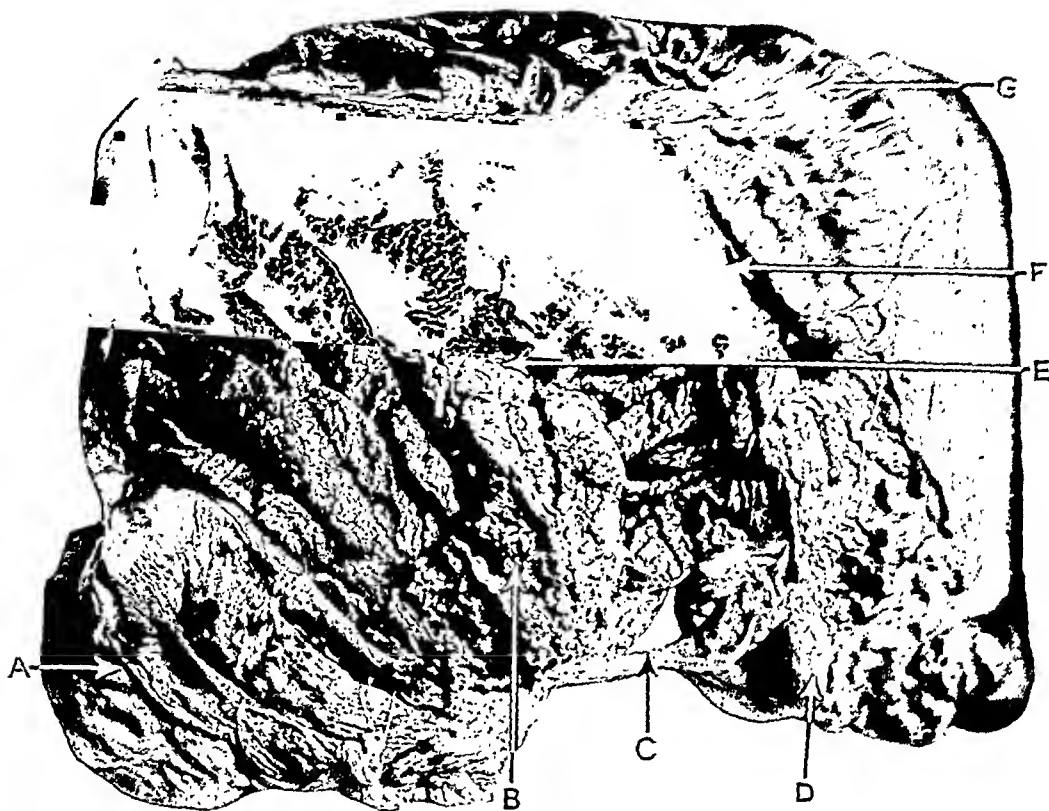


FIG. 46. — Pseudomyxoma peritonei arising from appendix (seen from behind). A, Sigmoid colon; B, Tip of appendix in mass of inflammatory tissue inside cyst; C, Appendix; D, Cæcum; E, Much corrugated lining, coated with yellow grit; F, Cut wall of cyst; G, Region of hepatic flexure.

gelatinous contents of the cyst gave a positive mucin reaction with *thionin*. Microscopically it was almost structureless, except for a little *very delicate* connective tissue, especially near the edges of a mass.

ON THE VEINS OF THE DIPLOË.

By GEOFFREY JEFFERSON,

HONORARY NEUROLOGICAL SURGEON TO THE MANCHESTER ROYAL INFIRMARY;
HONORARY SURGEON TO THE SALFORD ROYAL HOSPITAL;

AND D. STEWART,

LECTURER IN ANATOMY IN THE UNIVERSITY OF MANCHESTER.

THE existence of venous channels in the interior of the various bones of the human skeleton is to-day so widely, so universally recognized, that it seems strange that any era of anatomical knowledge can have existed when men went in ignorance of them. That the bones were well supplied with blood was certainly known, as the operation of amputation must have early taught the surgeon-anatomist. But that there were definite venous channels, that ordered systems existed in the interior of the bone, and particularly that these were present in the diploë and vertebræ, were certainly unknown until the earliest years of the nineteenth century.

In the year 1803 Dupuytren published in Paris a small monograph the first chapter of which is entitled "Canaux Veineux des Os." In this section he speaks of the little-known veins of the diploë as difficult or impossible to inject, but possible of display by section of the bone (as in the vertebræ) or by chiselling away the compact outer layer (as in the skull). Dupuytren gave a brief summary of the diploie veins, stating that there are three or four channels in the bone on each side draining from the summit downwards, and emptying into three sets of veins, namely, the external veins, the veins accompanying the meningeal arteries, and the meningeal sinuses of the cranial base. There is very little detail, and it is evident that Dupuytren is satisfied to be placing on record the bare outline of a newly discovered fact.

Here the matter rested until four years later, when Chaussier took up the anatomy of the diploie veins anew in his book (*Exposition sommaire de la Structure et des différentes Parties de l'Encéphale ou Cerveau*). This author gives an admirable description of the vessels, and accompanies it with a single clear explanatory plate. But he makes no mention of Dupuytren, and, indeed, so far is he from crediting Dupuytren with their discovery that he states on p. 18 of his preface that the diploie veins "have been discovered by M. Fleury, prosecutor of the École de Médecine, and now doctor and surgeon-in-chief of the Hospital of Clermon-Ferrant." These bibliographical points are interesting and rather confusing, and our confusion is increased when we find that anatomical text-books to-day almost all attribute the veins to yet another person, the distinguished French anatomist Gilbert Breschet. It was not until 1819 that anything on these veins came from Breschet's pen, the first of a series of three papers, each of increasing importance, and culminating

in the magnificent volume of studies which began to appear in 1830 and was never completed. The solution of the problem as to priority is to be found in a paper by Raciborski, "*Histoire des Découvertes relatives au Système Veineux*," 1836, from which we gather that Chaussier was indeed correct when he attributed the discovery of the veins to Fleury. Raciborski refers the reader to a letter written by Fleury to the *Gazette Médicale* in 1836 in which Fleury states that he found the diploic veins by chance whilst preparing skulls for the museum. He removed the outer table to exhibit the diploë, but going a little deeper than usual came across the venous channels. He told this fact to his intimate friend Dupuytren, who made it the subject of a memoir read to the Société de l'École de Médecine, and later included it in the thesis of 1803 to which reference has already been made. Raciborski remarks, "If we regard Dupuytren as the first of the authors who have spoken of these venous channels in the bone, we ought always to render justice to M. Fleury and regard him as the author of this discovery." He refers also to the enmity

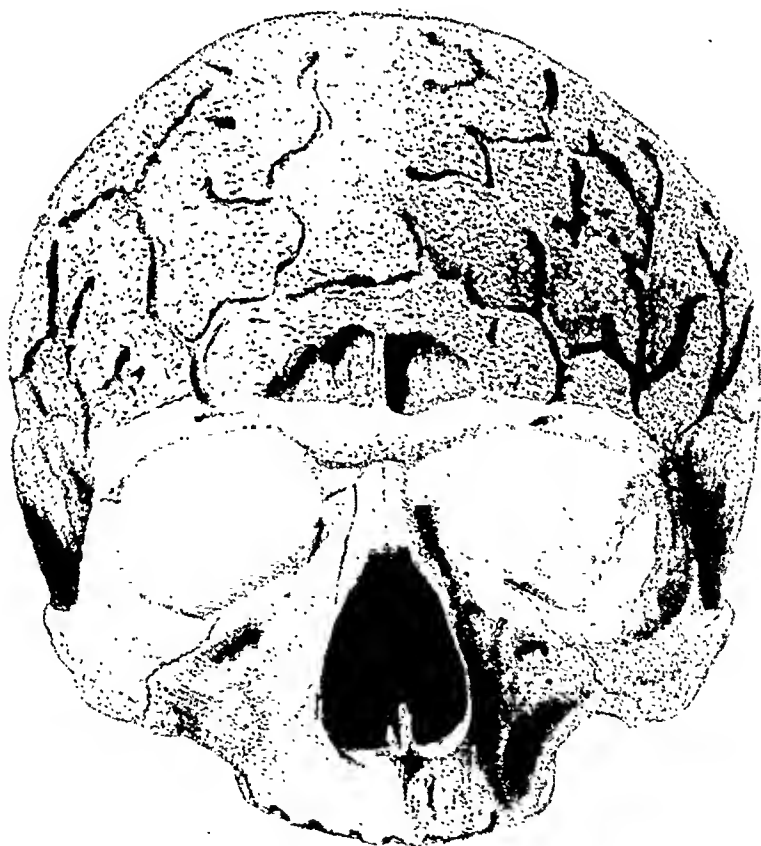


FIG. 47.—The frontal diploic veins. Note their emergence at the well-known emissary foramen in the supra-orbital margin (*Breschet*).



FIG. 48.—Lateral view to show the anterior and posterior temporal diploic veins and their connections (*Breschet*).



FIG. 49.—To show variations and communications with the meningeal veins (*Breschet*).

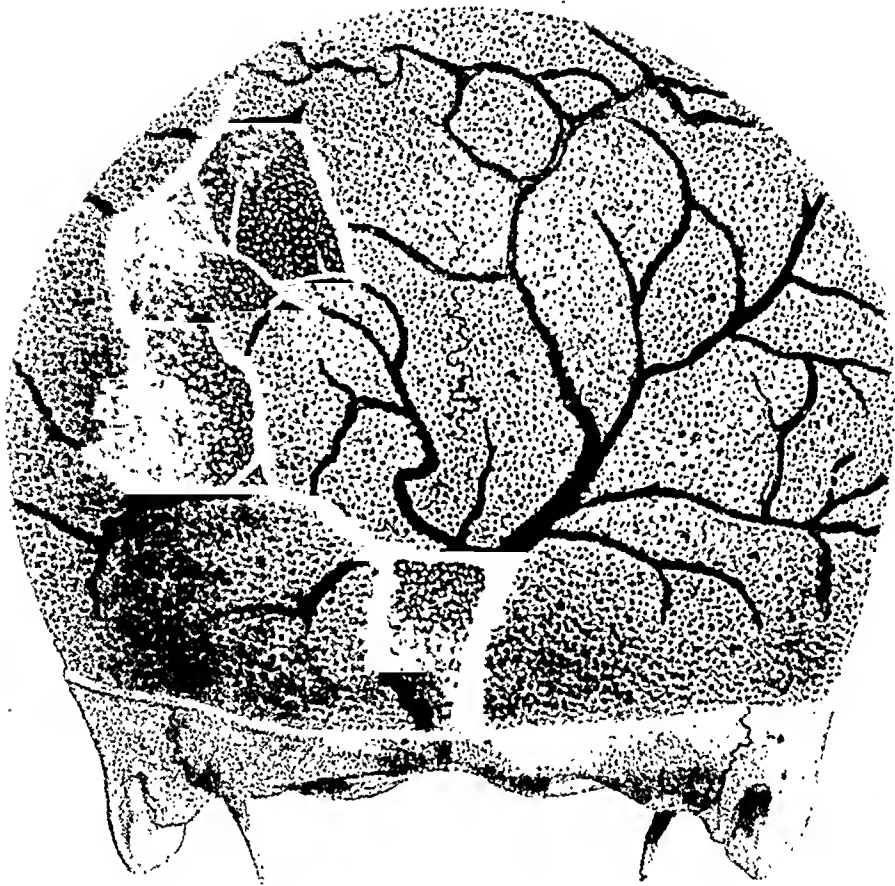


FIG. 50.—To show the dispositions of the occipital diploic veins, communicating with the posterior temporals or parietal veins (*Breschet*).

between Chaussier and Dupuytren, whence the omission of any reference by the former to the early paper on the "canaux veineux des os," and his particularizing Fleury, in his preface, as the real discoverer.

Mention has already been made of Breschet's papers. In 1819 he gave to the world his masterly exposition of the veins of the vertebral column, and made a passing reference to the veins of the diploë in speaking of intra-osseous venous channels in general. He returned to the diploic veins again in 1826, when he described them in some detail in a paper in German ("Nova Acta Physico-Medica, Naturæ Curiosorum," Bonn). He describes their development from half-canales at the centres of ossification of the principal bones (this, in passing, the present writers have confirmed). He also describes the minute pitting of the walls of these canals, proving that they are fed by innumerable venules from the meshes of the diploë.

Breschet's real claim, in popular estimation, to association with the diploic vessels, rests without question on the publication in 1830 of a superb volume, *Recherches Anatomiques, Physiologiques, et Pathologiques sur les Canaux Veineux des Os*. This beautifully illustrated work was an ambitious undertaking, and Breschet did not live to see it completed. It is an ironical fact that although there are twelve chromolithographic studies of the veins (four of which we are able to reproduce, by courtesy of the Librarian of the Royal College of Surgeons), there is no description of them in the text, no doubt owing to the unfinished state of the monograph. But judging from the plates it is evident that Breschet's studies were very complete, and had been carried further since his German publication. His account of the spinal veins has never been surpassed, and remains the standard description to this day. No doubt the diploic veins would in time have been equally described; in any case the beauty of the plates and their self-explanatory clearness has compensated for lack of text, and caused his name to be associated by a later age with this system of calvarial veins. In this posterity has unwittingly given him greater credit for originality than was his due, but knowledge advances step by step, and the final embellishment may be worth more than the bare outline of a first observation.

Forty years later Trollard, in a *Thèse de Paris* ("Recherches sur l'Anatomie du Système Veineux du Crâne et de l'Encéphale"), enters into a new description of the veins. He dissociates himself from the classical descriptions of Chaussier and Breschet, believing that the diploë is a spongy tissue with areolæ full of blood. The walls, according to him, are absorbed as age advances, and thus irregular channels are formed, but no definite or constant venous trunks. The earlier writers had believed that the vessels which they could easily trace with the naked eye arose from small radicles. This belief Trollard also combats, setting up in its place the idea that the veins start in blunt lacunæ, acquiring at once their full width. This point is worth mentioning, because on rough dissection the veins do indeed often appear to commence in this wise. We shall see, however, that this conception is incorrect. All veins save the largest trunks do notoriously vary greatly, and, so far as concerns this point alone, Trollard may have had some justification for his views. But modern injections and X rays prove that he was fundamentally wrong.

Another French monograph, one by E. Boismorreau, deserves something more, perhaps, than passing reference. In 1904 he published his inaugural thesis, "Contribution à l'Étude de la Vascularisation du Diploë," and gives a very sober and well-documented description of the diploë and its vessels, confirming the views of the earlier writers as against those of Trollard.

One last paper calls for comment. Elsberg and Schwartz have described the radiographical appearances of these veins with special reference to meningeal endotheliomas, and have given them a certain significance. Our own observations show that the veins may be well seen in perfectly healthy persons, whilst in a large series of tumour cases only occasionally was it possible to establish any notable increase in the size or field of distribution of the veins.

THE NORMAL VASCULARIZATION OF THE BONES OF THE CRANIAL VAULT.

The worker eager for information on the normal means of vascularization of the cranial vault will find very little to help him in readily accessible literature. It is evident that the flat bones of the skull, having no epiphyses, and a natural history of growth and function which differs widely from the long bones of the limbs, will have a blood-supply differing quite distinctly. In the long bones there is a system of nutrient arteries, one (or more) of which enters the shaft and divides to run to the opposite ends of the medullary cavity, where it anastomoses with the metaphysal sets. These nutrient arteries, despite the somewhat large openings in the cortex of the bone, are usually surprisingly small when studied by opaque injection and X rays. The calvarium, on the other hand, presents a different picture. In the place of the single nutrient foramen there are a number of small openings, limited to well-defined areas, the sites of which differ materially on the inner and outer surfaces. The outer table is generally smooth and very faintly pitted, but no openings large enough to admit a needle point will be found except in the areas of muscular attachment, as in the anterior part of the temporal fossa, and particularly in the occipital region below the superior curved line, where a variably large number will be found. These give entrance to branches of the muscular arteries which pierce the bone for its supply, fine vessels with a short course, making up in number what they lack in size. The foramina equally give exit to diploic veins. It is a commonplace experience of cerebellar surgical approach to meet with free venous oozing below the fringe of muscular attachment as the muscles are swept off the occipital bone. The divided vessels are apt to be loosely referred to by the operator as efferents of the lateral sinuses and torcula. More often they are not true emissaries, that is, freely perforating vessels, but are diploic efferents. This is clearly demonstrated by the behaviour of the troublesome bleeding points often encountered about the external occipital crest, for bleeding from them is easily stopped with wax, and when the bone is rongueured away the dural surface beneath is smooth and dry.

Apart from diploic connectors of this kind there are no noteworthy openings on the cranial vault save those of the well-known symmetrical emissaries, the mastoid, parietal, and so forth. Careful study of these latter foramina will frequently show that as they pierce the bone they are joined by diploic veins, a small perforation or two being discoverable in their smooth walls.

The arrangement of nutrient vessels is quite different on the inner surface of the skull, which, apart from the irregularities due to convolitional marking, presents the characteristic meningeal vessel grooves. On this surface again we find the multiple foramina which we saw at certain points on the external table, and once more they are strictly limited in their distribution; but now they occur only in the meningeal grooves and in and about the grooves of the great dural venous sinuses, a fact which gives us a clear lead as to the function of the meningeal vessels.

The Meningeal Arteries.—The meningeal arteries, and particularly the middle, are so hallowed by association with a clinical syndrome, that the reason for their presence may often escape observation. We may well ask ourselves why a fibrous membrane which should require no more vascularization for its own needs than the fascia lata of the thigh should be so richly supplied with vessels. For the dura is indeed very freely supplied with vessels—vessels so small and so easily controlled at operation that one is apt to be misled as to their number. We must confess that, with preconceived views formed at operation, injected specimens of dura came as a surprise;

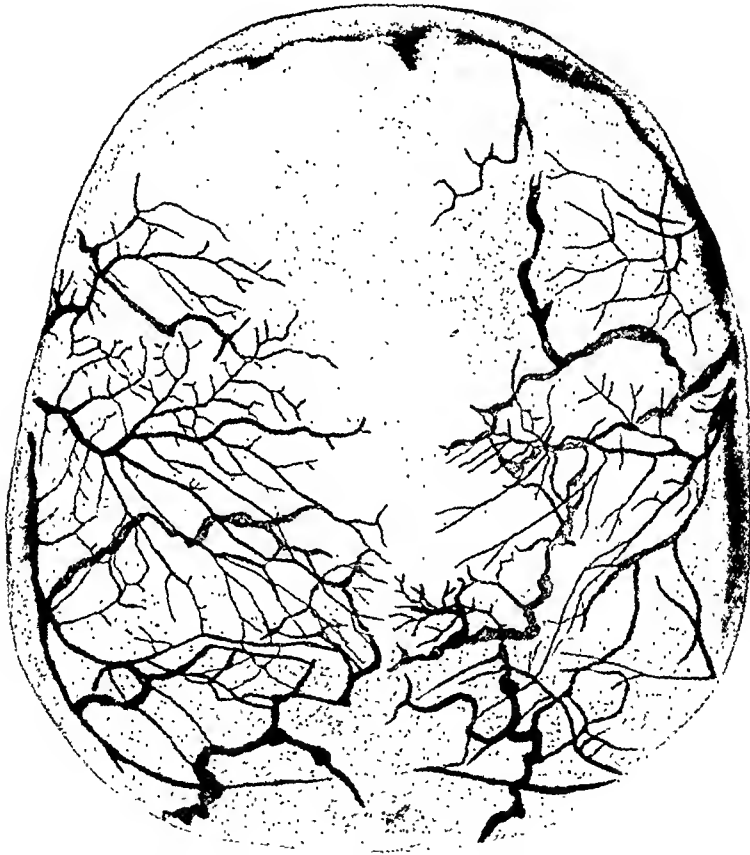


FIG. 51.—Radiograms of calvarium of dry skull. Diploic vessels in blue, meningeals in red.

many fine branches will be seen ramifying up to and beyond the sagittal sinus. The injections confirm the absence of any special meningeal arterial supply to the Paechionian bodies. But from our point of view, at the moment, the small puffs of lipiodol seen along the vessel and at the ends of the twigs of the arterial tree are of more importance. These represent the points at which these fine vessels were entering the bone as nutrients. When the artery, on such a specimen, is washed out with water, small pools collect at many points for the same reason. Conversely, when water

is forced into any channel in the sawn edge of a removed calvarium at post-mortem the inner table is seen to 'weep' in like manner. It is quite evident, then, that the meningeal vessels are not destined for the supply of that membrane alone, but rather for the overlying bone, of which the outer layer of the dura has long been known as the inner periosteum. At the point where the middle meningeal vessels tunnel the greater wing of the sphenoid, opportunity is taken for the interchange of vessels with the interior of the bone, venous connections being made here in particular,

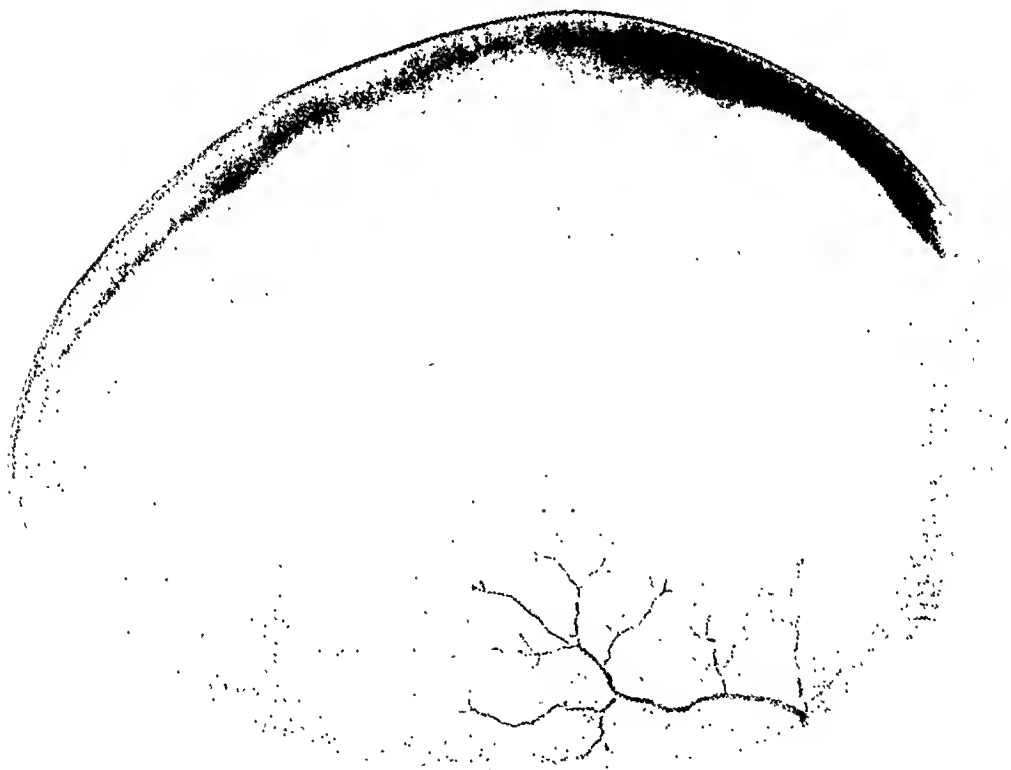


FIG. 52.—Lipiodol injection of the meningeal vessels in the bone.

though anastomoses occur besides at all points along the course of the vessels.

Meningeal Grooves.—It will be admitted by most who have worked at the subject that Wood Jones was correct when he re-established the old teaching and assigned to the veins the chief part in the formation of the meningeal grooves. So often at post-mortem one finds a deeply-cut groove running up to the parasagittal sinus and Pacchionian markings, whilst the artery supposed to have caused the bony track is seen tapering away until it is so thin above as to have been quite inadequate to occupy the wide furrow. It is a fact that the middle meningeal vessels, and their grooves, make their way up to the Pacchionian bodies, essentially venous structures. There is a capillary system at the origin of these veins, for on section in the living there

is not that severe reflux bleeding that would occur if they connected directly with the sinus.

The Vascularization of the Calvarium.—In summary it may be said that the bones of the vault are supplied with blood by branches of the meningeal vessels, and particularly the middle meningeal; that these branches penetrate the inner table and are generally of small size. Reinforcement is effected by arteries which enter the outer table, particularly in the areas of muscular attachment. But the larger channels in the bone are, as we shall see, essentially venous, an anatomical corroboration of another operative observation—that bleeding from the cut or nibbled calvarium is predominately venous in type. The vascularization of the cranial bones may be regarded as a seepage from very numerous vessels. It seems probable that this is not a unique method of circulation in bone, for the supply of the ends of the long bones and the compact tissue is of a rather similar type. In the cranium it would appear that the meningeal arteries replace the long nutrient arteries of the limbs, the extra-osseous course of the former being an exigence of different bony function and structure. Now it is a well-accepted fact that an osteoplastic flap may be lifted without fear of necrosis of the bone, so long as it remains attached to the temporal muscles: adhesion to the overlying skin is not essential. There is no question that at times replaced bone-flaps must approach the category of free grafts; and it is equally a fact that porosis of flaps does at times occur, though rarely. These bone segments are free of the dura when the bone is reflected and the fine vessels entering from this side are torn off. This deprivation is, as has been said, not a serious matter, so that whilst one may truly lay it down that the meningeal vessels are largely for the use of bone rather than of the fibrous matrix in which they lie, they are not in themselves essential for the continued life of that bone. Not only will the bone survive, but it is at times re-united by bridges of new bone. Union may be expected more often in the young, though we have seen it in older people; there seems to be no rule on this point. A flap a year old in a man of 30 was recently re-turned and fibrous union only was found; in another, re-opened after nine months (male, age 42), there was firm bony union along one border but not elsewhere; in yet a third (male, age 30) the flap six years later was found to have re-united in almost all of its circumference, though a groove marked the position of the original section.

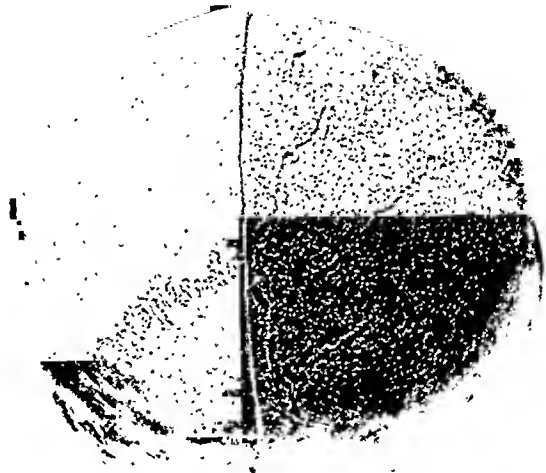


FIG. 53.—The occipital veins of the diploe. (From a specimen in the Museum of the Royal College of Surgeons, Edinburgh. Note the depth from the surface.)

THE ARRANGEMENT OF THE DIPLOIC VEINS.

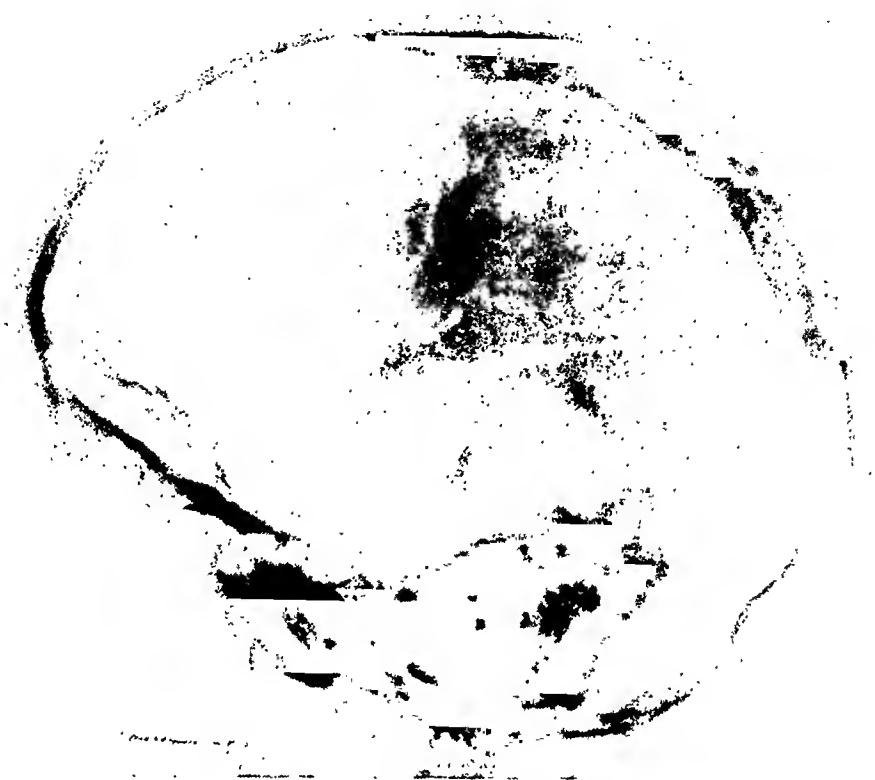


FIG. 54.—Diploic veins from a normal man of 55.

Breschet's own figures can scarcely be bettered, so we have chosen to illustrate this note with four of them (*Figs. 47-50*). They are so clear that little explanation is needed. As with venous channels elsewhere, considerable variation in size and position is to be expected. They lie nearer the inner than the outer table, and whilst each of the bones of the vault seems to have a set particularly of its own, connecting branches cross the sutures to unite the various groups. The veins, as seen in skiagrams of the living, group themselves into three main sets, frontal, parietal, and occipital. A V-shaped arrangement with the apex downwards is very characteristic (*see Figs. 47, 48, 50*), especially in the frontal region. The exit of these frontal veins by a small foramen in or about the supra-orbital notch is clearly seen in most dried skulls. The occipital set drain into large channels, one on either side of the mid-line, converging towards the base. The much larger parietal set includes the anterior and posterior temporals described by Breschet and others. Of the two, the posterior group are those most commonly seen in X-ray photographs of the living, and connections with the occipital diploic vessels are common, whilst the anterior temporal diploies are hard to see, for confusion arises between them and the grooves of the meningeal vessels,

with which they are closely connected. *Fig. 50*, the skull of an old man, shows actual defects along the course of this vein. Examination of the meningeal grooves in the inner table of the aged reveals, not the fine pitting of younger skulls, but many places where the floor of the groove is missing to a greater or lesser extent. Here the two systems become confluent.

It is important to recognize that this anastomosis takes place between the diploic channels and both the anterior and posterior divisions of the middle meningeal, for on the skiagrams of living skulls it is not uncommon to see the meningeal grooves run back and up, to be lost towards their termination in a typically diploic constellation.

We have confirmed the course and distribution of the diploic veins by the study of X-ray photographs both of the living and of dry skulls. Three attempts at injection on the cadaver with Ferguson's mass did not give the results expected. In the dry skulls the specimens used were either calvaria which had been separated from the remainder of the skull by a horizontal cut, or lateral halves of skulls which had been divided in the sagittal plane. By using these it was possible to avoid the images of the veins of the two sides being superimposed on the film. It had, however, the disadvantage, in the specimens of the calvaria, that only the superior portions of the veins could be seen.

When the X-ray films of the dry skulls (*Fig. 51*) were examined and compared with the dural aspects of the skulls themselves, it was found that not only were the grooves for the meningeal vessels shown on the film, but also other impressions obviously made by other blood-vessels. These were the diploic veins. It is not a difficult matter to distinguish the diploic from the meningeal channels without examining the inner aspect of the skull, as the former have certain characteristics: (1) While the general course of the meningeal vessels is upwards and backwards from the region of the small wing of the sphenoid, the diploic veins often run at right angles to this direction (especially in the posterior parietal region). (2) The origin of the larger diploic veins tends to be blunt. (3) There is a curious star-like branching and anastomosing of veins, which we term the 'spider' (*Figs. 56-59*). This is usually best seen in the parietal region, but may occur elsewhere. (4) The Y-like branching of the veins, commonly with short thick limbs, may be clearly made out.

When skiagrams are taken of the living skull, the results are complicated by the superimposition of the grooves of one side upon those of the opposite side, when seen in a film. The diploic markings, however, have the same characteristics as seen in the dry skull, which renders it comparatively easy with practice to distinguish the diploic veins from the meningeal vessels. The edges of the diploic grooves are quite sharply defined, but frequently show a recognizable crenation.

In the film of the dry skull, although it was quite simple to trace out the course of the main diploic vessels, it was difficult to differentiate the finer grooves by simple comparison of the skull and the film. To overcome this difficulty the following technique was adopted. On a matt print of a film all the larger meningeal grooves, as observed on the inner surface of the skull, were painted in red, and then by comparing the skull and the film all the larger

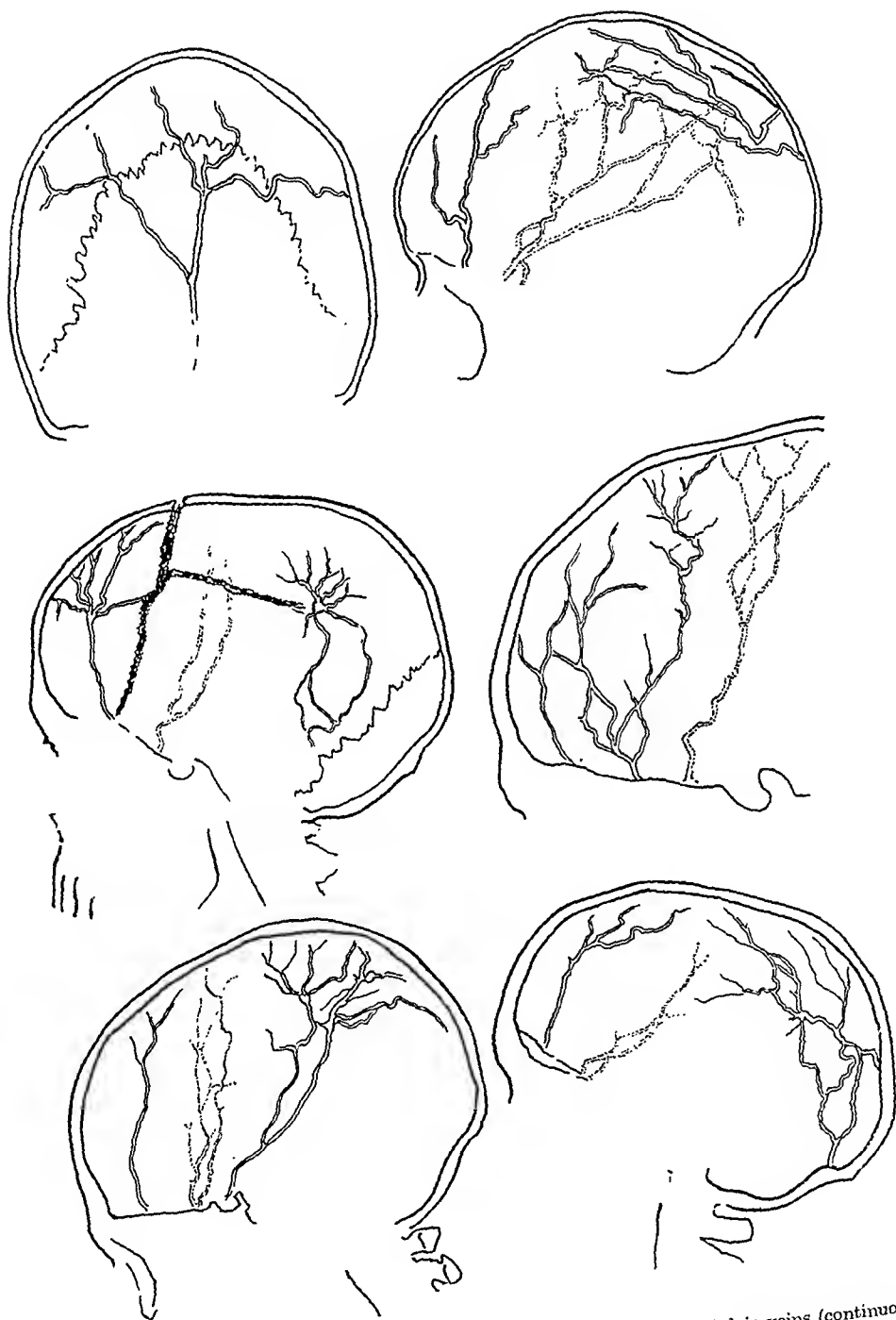


FIG. 55.—Drawing from X-ray photographs of variations in the diploic veins (continuous lines) and meningeal vessels (dotted lines). In the third there is a fracture; the horizontal limb was reported as ending in a "stellate crack."

DIPLOIC VEINS SHOWING THE 'PARIETAL SPIDER'.

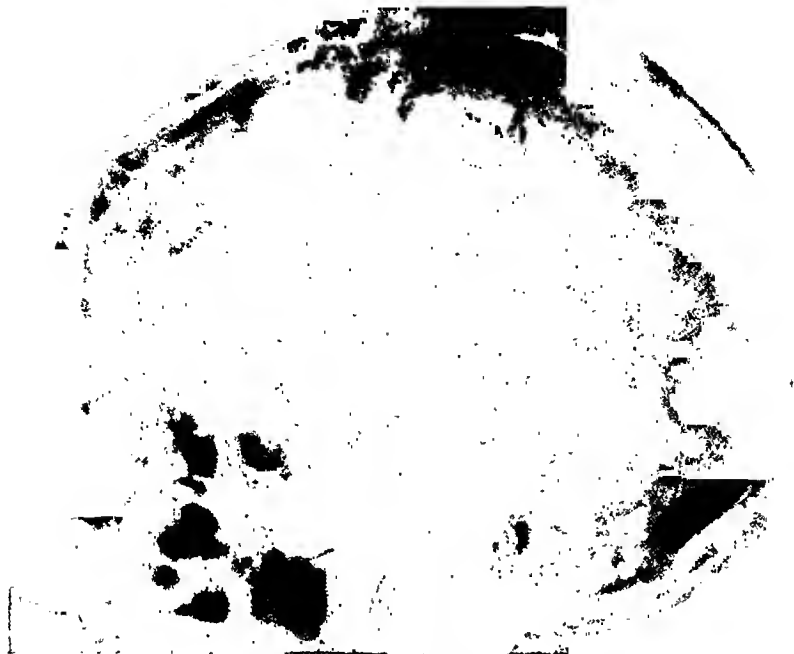


FIG. 56.—A male, age 24.



FIG. 57.—A male, age 28.

DIPLOIC VEINS SHOWING THE 'PARIETAL SPIDER'—*continued.*



FIG. 58.—A male, age 48.



FIG. 59.—A female, age 50.

diploic grooves were painted in blue. Then by a careful examination of the film, which showed more details than the print, it was possible to differentiate and paint in many of the smaller vessels. *Fig. 51* shows a print dealt with in this way, and gives a very clear idea of the distribution of the diploic veins. The results obtained by the methods described above generally confirm the findings of the older observers. The frontal, posterior, temporal, and occipital veins were easily traced, and it was found that the posterior parietal and the occipital constantly and freely anastomosed with each other in the parietal region, forming the 'spider.' The frontal diploic veins were more isolated, and connections could only rarely be traced between them and



FIG. 60.—Diploic veins in a case of adult hydrocephalus of long standing showing the lakes of confluence.

the other veins. It will be noticed that no mention has been made of the anterior temporal vein. This is due to the fact that it was not seen in the dry skulls and cannot often be unequivocally distinguished in the living. Its place frequently appears to be taken by a vessel grooving the inner aspect of the skull in front of the middle meningeal grooves, which frequently originates in a depression near to the great longitudinal sinus; it ends in the sphenoparietal sinus. We are of the opinion that this vein may be the anterior temporal diploic vein placed more deeply in the dura than the other diploic vessels, which are placed more superficially in the bone.

Its fate seems therefore to be bound up with that of the anterior division of the middle meningeal vessels. *Fig. 60* is from an old-standing hydrocephalus (female, age 24). The sharp-cut groove for the middle meningeal vessels is replaced by a chain of small pools of blood. This figure may be most usefully compared with Brechet's figure (*Fig. 49*). (This possible interchange has been noticed by Coen, and is referred to in a paper by Thompson, which we have seen only after the above was written). The very deep markings of the meningeal grooves figured by Elsberg and Schwartz may have a similar genesis.

CLINICAL APPLICATIONS.

In surgical practice the veins of the diploë will most often cause confusion in X-ray cases of head injury (*Fig. 61*). It was, indeed, the report of a radiographer that extensive stellate fractures were present in the parietal region of two patients who had had no injury at all, that drew the attention of one of us to the problem of the calvarial vessels. Generally a fracture line is easy to



FIG. 61.—X-ray showing diploic veins on the right, and a fracture cutting the left orbital margin and simulating a diploic vein. The line of fracture is shown by the arrows.

distinguish owing to its straightness, its clear-cut margins, and its extent. The great safeguard is an adequate knowledge of the normal appearances of the veins, their position and direction, their tendency to anastomose and form 'spiders,' stars, and Y-junctions. We have found clinically that the most difficult tracks to assess are those parallel with the contour of the skull high in the parietal region. It must be remembered that the veins, like

fractures, often run across suture lines, but anyone familiar with them will hardly fail to distinguish them from injuries. A mistake in a case involving a civil action at law, as in the now common motor accident, may be costly. Further, it is possible that deep venous erosions may assist in determining the line of fracture, as Elliot Smith showed years ago and as Thompson again suggests.

A dilatation of the veins in the diploë might be thought to be a likely concomitant of intracranial tumour, with its pressure on the venous side. We have referred to the serious venous oozing met with at certain times and in certain places, and emphasized that the bleeding is commonly from diploic efferents as well as from perforating emissaries. Now it is quite true that the eye skilled in the detection of the diploic channels in skiagrams of the living may frequently note here an unusual clarity in the vessels' lines, there an uncommon pool of blood. Yet the fact remains that the most beautiful examples of diploic veins that we have seen have been discovered by chance in skiagrams of non-tumour cases. The observer would therefore be very wrong if he concluded that because the veins were very distinct a tumour was present in the skull. The age factor has been remarked on. In children with their sparse diploë the veins are small and hard to define; as age advances they become clearer. When the skull is thinned by expansion in hydrocephalus above infancy the veins are again ill-seen, save that around the great meningeal collecting trunks diploic markings become more evident (*Fig. 60*). But it is impossible to lay down a rule that shall be true of every case; some will demonstrate one point and not another. There seems to be some ground for the views of older writers, that the diploic system acts as an additional reservoir for the blood circulating within the cranium and might act as an escape or balancing mechanism when that current is disturbed. Operative experiences with tumour cases support this belief.

In meningioma involving the overlying bone there is no question as to the increased diploic bleeding, clearly localized as to its source by the uniform failure of ligature of the external carotid artery to abate it. It is evident that the time factor must be very important, and that if diploic irrigation is to be increased in a tumour-bearer a considerable period must elapse before dilatation recognizable on an X-ray film can occur. We can only repeat that inferences drawn from the radiological appearances of these veins without reference to other more important clinical factors will almost certainly be faulty.

One last word, and that on infection. Acute pyogenic osteomyelitis is rare in the vault owing to the absence of epiphysial lines. On the other hand, the venous channels allow of easy spread from one area to another, and this explains the patchy distribution of calvarial infection when it does occur. A case recently observed will serve as illustration. A young man began with a furuncle on the nose which rapidly caused œdema and cellulitis in the orbit. After a stormy convalescence a deep temporal abscess formed. Later patches of osteomyelitis developed in the frontal and high in the parietal region. Infection could easily find its way by means of a septic thrombophlebitis originating in the deep veins of the temporal fossa and spreading by way of the vessels, which enter and leave the bone in this region, with septic permeation and osteomyelitis at a distance.

SUMMARY.

The discoveries of our forefathers have a habit of attaining a new importance many decades later. This is exemplified by the veins of the cranial diploë, which refinements of X-ray technique now render clearly visible in most radiograms of the living skull. These veins, which occupy very definite channels in the substance of the bone, may be easily mistaken by the unwary for fractures. Generalized enlargement, detectable by radiography, is not the rule in cerebral tumour, and beautiful examples may be met with in normal persons, especially in the later decades of life. Unusual clarity and breadth of the middle meningeal grooves is more often met with in cases of intracranial tumour. In these cases the anterior temporal diploic veins add their quota to the impress of the meningeal vessels. The meningeal vessels, both arteries and veins, are destined to a considerable extent to supply the bone.

Figs. 47 to 50 are photographs from Breschet's book made by kind permission of Mr. V. G. Plarr, Librarian to the Royal College of Surgeons. *Figs. 51, 55, and 60* are by Miss Davison. We are indebted to Mr. Greig for *Fig. 53*. We have to acknowledge also the interest which Mr. Higgins, Radiologist to the Salford Royal Hospital, has taken in the work, and are indebted to Dr. Morris, of the same department, for the prints of *Figs. 56-59*. Professor Stopford has been most kind (as ever) in allowing the free use of material in the Department of Anatomy.

BIBLIOGRAPHY.

- DUPUYTREN, G., *Propositions sur quelques Points d'Anatomie, de Physiologie, et d'Anatomie*, 1803.
- , *Sommaire de la Structure et des Différents Parties de l'Encéphale ou Cerveau*, Paris, 1807.
- BRESCHET, G., *Essai sur les Veines du Rachis*, Paris, 1819.
- BRESCHET, G., "Anatomisch-Physiologische Untersuchungen über einige neu entdeckte Theile des Venen-systems", *Nova Acta Physico-Medica*, Acad.-Caes. Leopold-Carol., Naturae Curiosorum, Bonn. Tom. dec., 1826.
- BRESCHET, G., *Recherches Anatomiques, Physiologiques, et Pathologiques sur le Système veineux, et spécialement sur les Canaux Veineux des Os*, Paris, 1830.
- RACIBORSKI, A., *Histoire des Découvertes relatives au Système Veineux*, Paris, 1841.
- TROLLARD, P., "Recherche sur l'Anatomie du Système veineux de l'Encéphale et du Crâne", *Thèse de Paris*, 1868, No. 256.
- BOISMORREAU, E., "Contribution à l'Étude de la Vascularisation du Diploë", *Thèse de Bordeaux*, 1904.
- SMITH, G. ELLIOT, "A note on Nervous Lesions Produced Mechanically by Atheromatous Arteries", *Rev. Neurol. and Psychiat.*, 1905, iii, 182.
- JONES, WOOD F., "On the Grooves upon the Ossa Parietalia commonly said to be caused by the Arteria Meningea Media", *Jour. Anat. and Physiol.*, 1912, xlvii, 228.
- ELSBURG, C., and SCHWARTZ, C. W., "Increased Cranial Vascularity in its Relation to Intracranial Disease, with special reference to Enlargement of the Veins of the Diploë and its Relation to the Endotheliomas", *Arch. of Neurol. and Psychiat.*, 1924, xi, 292.
- COEN, B., "A Communication as to the Causation of Large Vascular Grooves found on the Inner Aspect of the Ossa Parietalia", *Jour. Anat. and Physiol.*, 1914, xlviii, 293.
- THOMPSON, I. MACLAREN, "On Certain Grooves upon the Deep Aspect of the Cranial Vault", *Canad. Med. Assoc. Jour.*, 1926, Oct., 1194; "A Note concerning the Radiographic Differentiation between Diploic and Meningeal Vascular Channels", *Ibid.*, 1927, Jan., 64.

PSEUDOCOXALGIA : CLINICAL EVIDENCE, X-RAY APPEARANCE, ETIOLOGY, AND TREATMENT: WITH A REVIEW OF 25 CASES.

By C. LEE PATTISON,*

CONSULTING SURGEON TO THE TUBERCULOSIS DEPARTMENT, ROTHERHAM CORPORATION;
MEDICAL SUPERINTENDENT OF THE KING EDWARD VII HOSPITAL FOR CRIPPLED CHILDREN, SHEFFIELD.

SYMPTOMS AND PHYSICAL SIGNS.

FREQUENTLY the symptoms and signs of pseudocoxalgia are so indefinite at first that the disease is unnoticed in the earlier stages.

The most constant symptom during the onset of the disease is a limp: there is a tendency to drag the affected limb, the foot is not lifted freely from the ground, but is swung round in an everted position with each step. The limp is not painful and is frequently intermittent; for two or three weeks at a time it may not be present. After from six to eighteen months, when the disease has become more fully established, the limp frequently disappears, but if it persists it may change its character and resemble the 'pain' limp found in infective arthritis of the hip-joint. Pain is said to be absent in nearly half the cases, but it will be seen that it was present in the majority of my patients at their first examination. It may be slight and localized to the hip, or referred to the knee, while in a few cases it is extremely severe.

Some degree of muscle-spasm can usually be found during the early stages of the disease. As a rule it only lasts for a short time, but may recur, and often persists longer than the pain and tenderness. This will result in a painless limp, the limb being fixed in an adducted and slightly flexed position, in contrast to the abduction found in early tuberculosis.

The amount of movement possible in the joint depends largely on the degree of muscular spasm, and will therefore vary considerably. Frequently the movements are nearly perfect throughout the disease, and even if limited at first they soon improve; but usually there is some limitation of abduction and internal rotation, which often persists in these two directions throughout life. Sometimes the hip may be nearly completely fixed, usually in a position of adduction and slight flexion.

Muscular atrophy may be either hardly noticeable or well marked. The localized thickening of the soft tissues so frequently felt in tuberculosis of the hip-joint is not found in pseudocoxalgia. The great trochanter of the femur is prominent and projects laterally. Observers differ as to whether it is found at a higher level than normal; in my own cases no such alteration was noticed. Abscess formation never occurs in this condition.

Osteo-arthritic changes take place in a certain number of cases at a late stage, and result in pain, stiffness, and further limitation of movement.

* A paper read before the Sheffield Medico-Chirurgical Society, October, 1927.

From a consideration of the clinical evidence of pseudocoaxalgia it will be clear that it is often extremely difficult to distinguish the condition from epiphysial coxa vara, tuberculosis, and other diseases. In fact in some cases it is quite impossible to do so with certainty by ordinary physical examination, but as the radiographic appearances precede the clinical signs the diagnosis can usually be made by the X rays.

SKIAGRAPHIC APPEARANCES.

Changes in the Head of the Femur.—In a normal child the head of the femur is cartilage with a bony nucleus in its midst which throws a shadow in the skiagram. The extent of the cartilage can be estimated by noticing the size of the clear space between the nucleus and the acetabulum.

1. *Stage of Flattening.*—At first there is an increase in the density and a diminution in the size of the bony nucleus, making the head of the bone appear flattened. The size of the epiphysis remains unchanged, but the joint interval appears larger as less space is occupied by the bony shadow (*Fig. 62, p. 95*).

2. *Stage of Fragmentation.*—The bony nucleus next begins to show irregular calcification; parts show increased density, while others undergo rarefaction. This process continues until the bony nucleus appears broken up into fragments—areas where lime salts are markedly increased in amount, with intervals of decreased calcification between them (*Fig. 63, p. 95*). At this stage, being deprived of its bony support, the cartilage begins to give way; it loses its spherical shape and becomes spread out laterally. The process of fragmentation may be very little marked, or it may continue until the head of the bone has scarcely any bony nucleus left.

3. *Stage of Repair.*—It should be remembered that the cartilaginous envelope is deformed; it is flattened and squashed out laterally, so that it envelops the circumference of the acetabulum, and when the bony nucleus re-forms, its shape is limited by the boundaries of its cartilaginous surroundings. The fragments begin to coalesce; then the density of the whole epiphysis gradually diminishes until it becomes uniform and equal to that of the opposite side. Eventually it increases in size until it nearly fills its flattened cartilaginous envelope (*Figs. 65–70, p. 95*).

4. *Stage of Moulding.*—The re-formed femoral head undergoes moulding until adult life. It is said that one of two types of head results, either (a) a globular, round head, or (b) an oval head. Legg² calls these the 'mushroom' and 'cap' types respectively, and it is generally thought that these two types are quite distinct, and that the function of the former is ultimately superior to that of the latter. In my experience it is often very difficult to know in which category to place many cases, for the line dividing the two types is frequently poorly defined. With regard to the ultimate degree of function, all that can be said is that the more the shape of the head differs from the normal the worse it will be. This will become more noticeable in later life, as in cases with deformed heads osteo-arthritic changes are likely to occur.

Changes in the Neck of the Femur.—The contour of the neck alters, its upper part becomes broader and its upper angle rounded off, while later on there is shortening of the neck as a whole, producing an apparent coxa vara.

During the beginning of this stage an ill-defined zone of change in calcification produces a sponge-like area in the upper part of the neck. This metaphysial change is early and constant (*Fig. 71*, p. 96). Later a regular pattern-like appearance is sometimes noticed; there are zones of increased calcification arranged as pockets opening towards the epiphysial line, each pocket enclosing a corresponding area of rarefaction. This phenomenon is said to be associated with fragmentation, and to disappear early during the period of healing, but in the few instances where I have noticed the appearance it has been quite a late occurrence.

Changes in the Acetabulum.—Opinions differ as to whether any changes are constantly found in the acetabulum; and whether these are primary if they do occur, or only late hypertrophic changes secondary to those in the head. Perthes³ says that the acetabulum only shows changes in advanced stages, while Sundt⁴ considers that the acetabulum is normal late in the disease. Platt,⁵ however, thinks that changes "can be seen at every stage, and that they should be considered partly as the adaptation of the cavity to the altered lines of pressure through the deformed head, and partly as of the same nature as the transformation undergone by the epiphysial nucleus and thus truly specific."

ETIOLOGY.

Age.—Pseudocoxalgia occurs most often between the ages of 5 and 10.

Frequency.—Calvé⁶ found 1 case in every 50 patients with hip disease. Gauvain⁷ reports an incidence of 1 in 24, while during the last five years the present writer has seen 25 cases among 360 patients with alleged tuberculosis of the hip-joint, a proportion of more than 1 in 15.

Sex.—Boys are more commonly affected than girls; most authorities consider the proportion to be about 4 to 1. Platt,⁵ however, in his 23 cases, found 12 boys and 11 girls. In the present series of 25 cases there were 18 boys and 8 girls.

Bilateral Cases.—These are comparatively few. Legg⁸ found 2 in 55 cases; Perthes,³ 2 in 23; Platt,⁵ 2 in 23; Sundt,⁴ 7 in 75; Pattison, 5 in 25.

Heredity.—Although a few cases have been reported in which the disease occurred in the same family, heredity does not seem to be a factor in the incidence of pseudocoxalgia. Neither syphilis nor rickets seems to be associated with the condition.

When investigating the cause of a non-fatal, chronic disease such as pseudocoxalgia, we are handicapped for lack of material for pathological examination. A number of surgeons have, however, operated on patients with this condition, either to determine the nature of the disease or in an attempt to remove the focus and so hasten the cure. It may be well to consider what has been found by exploration before examining the theories advanced to explain the pathogenesis of the condition.

The synovial membrane has sometimes been normal in appearance, but in a few cases it was thickened, showing evidence of mild synovitis; in these cases the joint-cavity was filled with a slightly turbid and straw-coloured fluid.⁹ No bacterial cultures have ever been obtained from the synovial fluid; nor has inoculation of guinea-pigs with the fluid shown evidence of bacterial infection.

Perthes³ removed a small piece of the head of the femur. The interior of the epiphysis showed replacement of the cancellous tissue by invading buds of cartilage; he considered the condition to be non-inflammatory. Legg⁸ scraped out a large area of rarefaction in the neck of the femur and grew staphylococci from the débris so obtained. These staphylococci were very scanty and may have been due to contamination. Kidner¹⁰ and McWhorter¹¹ also obtained isolated colonies of staphylococci from the granulation tissue found in necrotic areas in the metaphysial region. Phemister¹² removed part of the epiphysis after eurenting the interior. He found that the joint-cavity showed evidence of acute synovitis, although the articular surface of the deformed head retained its normal sheen. He obtained no microbial growth from the material, but observed changes which he considered typical of an old infective lesion of the bone, and probably of pyogenic origin.

None of the many theories which have been brought forward to account for the pathogenesis of pseudocoxalgia is alone adequate to explain the condition. A few of the most reasonable theories will be briefly considered.

The Infective Theory.—The exponents of this consider that pseudocoxalgia is produced by infective agents of low virulence. It has many supporters and has been ably upheld by Platt.⁵ He states that the condition is often associated with pyrexia, enlarged tonsils, or chronic ear disease, and often with a certain amount of ill-health. He holds the view that the disease is hæmatogenous in origin like other forms of osteomyelitis. He considers that the area of the femoral neck supplied by the upper leash of metaphysial vessels is that first affected, since that region is more vascular than the synovial membrane at the age when pseudocoxalgia is found. He explains the absence of the disease in older patients by the relative increase in the vascularity of the synovial membrane with age, which makes the joint proper more likely to suffer than the metaphysial region, and therefore the changes in the head we associate with pseudocoxalgia do not occur.

As we have already noticed, exploration of the joint has rather upheld the infective theory. Anyone who has seen cases of pseudocoxalgia in the active stage with marked pain, muscle-spasm, and limitation of movements, cannot help being impressed by the clinical resemblance to infective arthritis. It is well known that coxa plana occurs in lesions of the metaphysis known to be infections, e.g., tuberculosis.

Against the idea that the condition is due to an infection are these facts: abscesses never develop, there is no leucocytosis, the neighbouring structures are never affected, the radiographic appearances precede the clinical signs (contrast infected joints), and except in rare instances the femoral epiphysis is the only one affected.

Developmental Theories.—About the end of the second month of fetal life the human hip-joint resembles that of adult reptiles. There is an imperfectly formed and shallow acetabulum and a small malformed head. The limb is then at right angles to the axis of the body and rotated outwards, the extensor surface of the knee being directed upwards and the great toe in front. If rotation occurred at this time, to enable the erect position to be assumed, dislocation would be likely to take place.

Calot¹³ thinks that in pseudocoxalgia there is a condition of arrested

development of the hip-joint such as that just described, with a tendency to congenital dislocation of the hip, and that the result of this is that the head fits imperfectly into the acetabulum, with resulting osteochondritis from the mechanical disability.

Mürk Jansen¹⁴ has propounded a somewhat elaborate theory to account for the condition. He says that the curve of the acetabulum is flatter than the curve of the femoral head in patients who develop pseudocoxalgia, and that this is caused either: (1) By the bony floor of the acetabulum being too thick; or (2) By the ischium being bent inwards towards the mid-line, a condition of 'ischium varum'; and he states that these abnormalities are produced by a small amniotic sac which either tends to flex and adduct the hip-joint and lever the head out of the acetabulum and so lessen the normal pressure of the head against the acetabular floor; or else, by being tightly drawn over the buttock, bends the ischium inwards.

If the difference of curvature produced in either of these two ways be granted, Jansen says that the mechanical conditions which follow will naturally result in changes in the head of the femur.

The following objections have been raised to this theory:—

1. That ischium varum is seen if the radiograph be not taken exactly symmetrically, and that it occurs in many conditions without pseudocoxalgia following.

2. That in many cases pseudocoxalgia occurs without any discrepancy in the curvature of the head and acetabulum.

3. That if the condition were due to pressure on only a small part of the head, early cases treated by traction and fixation should be arrested.

In reply to this last objection, Jansen says that so long as any discrepancy of joint surfaces exists, abnormal stimuli will be acting, and that the measures of treatment adopted are insufficient to prevent this.

4. The theory has also been criticized on the grounds that pseudocoxalgia often occurs some years after the child has begun to walk. Jansen, however, asks us to consider other deformities, such as genu valgum, which also take time to manifest themselves.

The Traumatic Theory.—The epiphysis of the upper end of the femur is almost entirely dependent for its blood-supply on the small vessels entering its circumference where the head and neck join. At birth the upper epiphysis of the femur includes the great trochanter; later the neck of the femur develops as an upward extension of the shaft which eventually divides the epiphysis into two: (a) the head, (b) the great trochanter. This division is complete by the third year, which is the earliest age at which pseudocoxalgia has been noticed, and is the age at which there is for the first time great restriction in the blood-supply to the head. It is thought that a slight injury may result in obliteration of some of the vessels supplying the femoral epiphysis, with atrophy of bone-cells and decalcification. The cartilage, which gets its nutrition from the synovial fluid of the joint, remains unaffected; but with the disappearance of its bony nucleus the cartilage collapses; eventually, when the vascular supply is restored, new bone is laid down but the epiphysis is misshapen and deformed.

Recently Bentzon,¹⁵ by injecting alcohol round the arteries of the upper

epiphysis of the femur in rabbits and so paralysing the vasoconstrictor nerves, has produced coxa plana, which he considers is caused by the active hyperæmia so induced. He thinks that the arteries supplying the caput femoris are especially exposed to mild lesions in young people, where injury may bend the cartilaginous parts of the epiphysis.

Legg¹⁶ tunnelled into the neck of a femur for a definite acute septic condition; five years later flattening of the epiphysis occurred, due he considers to interference with the blood-supply during the operation.

TREATMENT.

There is a tendency towards spontaneous recovery in this condition, and patients never ultimately show complete incapacity; on the other hand, the end-results vary considerably so far as the function of the limb and comfort of the patient are concerned.

In the first case it is only fair to state that quite a number of observers consider that treatment has no effect on the condition. Legg² sums up the views of these surgeons when he says: "While a process suggesting weakness of bone structure is going on it is theoretically sound to allow no weight-bearing; but in practice, relief from weight-bearing in no way affects the end-result." The majority of surgeons, however, consider that if treatment be begun before the cartilaginous envelope of the epiphysis has been deformed, the subsequent malformation will be much less marked, although the fragmentation and cycle of bony changes will not be completely averted. An examination of my own cases bears out this view.

The treatment most commonly recommended is fixation in an abducted position, and measures to prevent the weight of the body being borne by the diseased limb. Some surgeons⁹ think that movement does no harm if the latter point be attended to. The duration of fixation necessary should be controlled by repeated X-ray examination to determine the degree of recalcification reached by the head of the bone, and may be any length of time between six months and three years.

In cases with a large amount of muscular spasm and pain, one is forced to treat the condition for the sake of the patients' comfort even if one has not much faith in its effect on the end-results. In these acute cases fixation is insufficient to relieve the pain, and some form of extension apparatus is necessary to overcome the muscular spasm.

Kidner¹⁰ and others¹¹ have operated on patients with this disease, on the assumption that it is a mild form of osteomyelitis. Kidner scraped out the débris from an area of rarefaction in the neck of the femur of a boy of 5. He observed by X rays the subsequent healing, and said that the operation hastened this and diminished the subsequent deformity. The great majority of surgeons, however, consider an operation of this sort to be unjustifiable.

Many years after the disease is apparently cured, pain and stiffness may develop. These cases may or may not show X-ray evidence of osteo-arthritis; in the latter instance operation to remodel the head may be desirable.

DETAILED REVIEW OF 16 CASES OUT OF 25 TREATED.

CASE 1.—Male. Age at onset, 6 years 7 months.



FIG. 62.

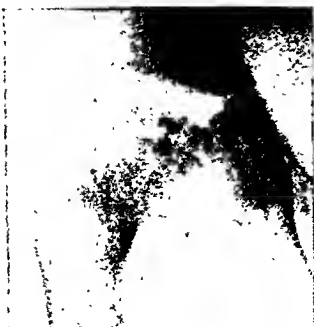


FIG. 63.



FIG. 64.

March 17, 1924 (Fig. 62).—Only movement possible was slight flexion. No pain. Metaphyseal rarefaction is well shown.

July 28, 1924 (Fig. 63).—Rotation, inversion, and abduction limited, other movements perfect. Treatment: weight-extension since admission.

Feb. 18, 1925.—Six months after discharge, without treatment, movements were perfect.

Jan. 26, 1926 (Fig. 64).—Seventeen months after discharge. No symptoms.

CASE 2.—Female. Age at onset, 3 years 4 months.



FIG. 65.



FIG. 66.

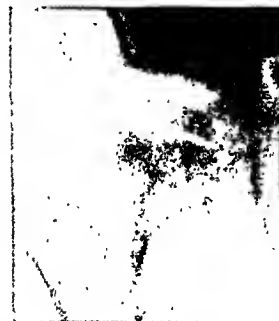


FIG. 67.



FIG. 68.



FIG. 69.



FIG. 70.

May 27, 1922 (*Fig. 65*).—Limp began 10 months before and has been intermittent. Now some pain and all movements limited, can walk with limp. During the 10 months since onset of first symptoms the head of the femur has practically disappeared.

Dec. 11, 1923 (*Fig. 66*).—Patient has been treated in bed for 7 months in a short hip-spica plaster. All movements except internal rotation and abduction are perfect; no pain. Patient was discharged from hospital with a light celluloid splint, crutches, and patten. Note metaphysial rarefaction.

May 5, 1924 (*Fig. 67*).—Movements nearly perfect. Splint removed, crutches and patten continued for further 6 months.

March 13, 1925 (<i>Fig. 68</i>).	} Further stages of repair. <i>Fig. 69</i> shows condition of head 2 years 10 months after treatment ceased.
Feb. 6, 1926 (<i>Fig. 69</i>).	
Sept. 8, 1927 (<i>Fig. 70</i>).	

CASE 3.—Male. Age at onset, 7 years 6 months.



FIG. 71.



FIG. 72.



FIG. 73.

Sept. 3, 1924 (*Fig. 71*).—Disease followed directly after measles 1 month ago. Attempt at movement in any direction causes considerable pain. Treated for 6 months with weight-extension and then for 3 months in a plaster splint. Note metaphysial rarefaction.

July 8, 1925.—Movements nearly perfect, no pain. No treatment except bed.

Dec. 10, 1925 (*Fig. 72*).—Discharged from hospital with no retentive apparatus. Movements perfect.

Aug. 4, 1927 (*Fig. 73*).—One year and 8 months after discharge from hospital. Movements perfect. Slight limp when tired.

CASE 4.—Male. Age at onset, 8 years 9 months.

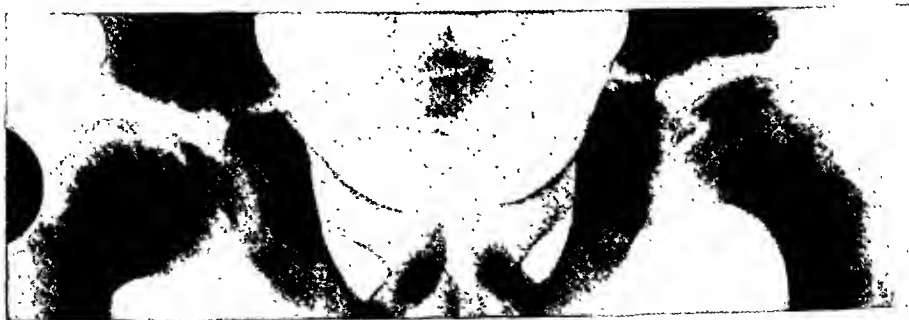


FIG. 74.

Dec. 30, 1926 (*Fig. 74*).—At first examination 6 months after onset of limp, had been treated as tuberculosis of hip. *Left hip*: very acute disease, marked pain, no movements possible. *Right hip*: no symptoms. Note metaphysial changes.

July 17, 1927 (*Fig. 75*).—Left hip: marked limitation of internal rotation and abduction. Marked limp, no pain.

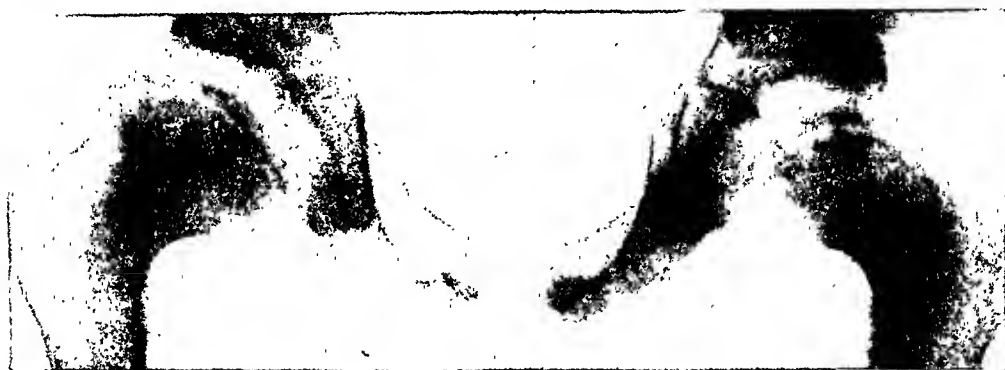


FIG. 75.

CASE 5.—Male. Age, 10 years.

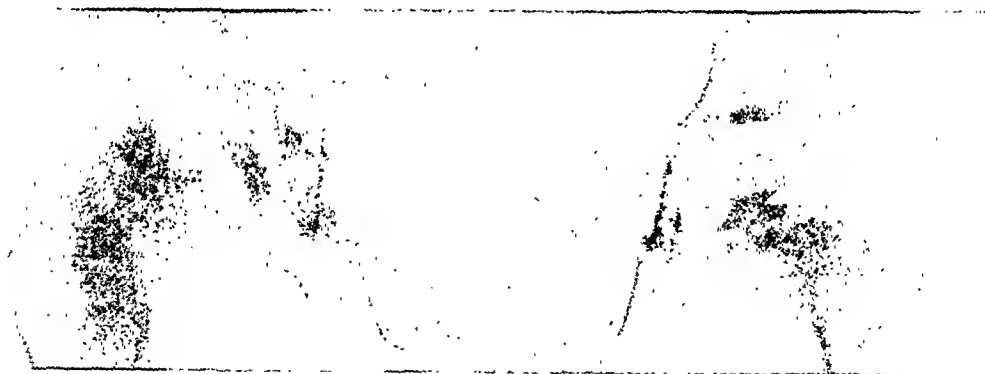


FIG. 76.

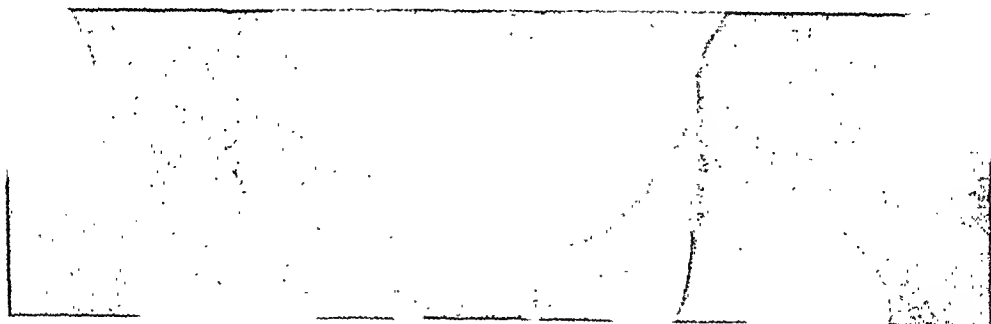


FIG. 77.

April 10, 1923 (*Fig. 76*).—Duration: right, unknown; left, 2 years. Right, no symptoms; left, slight muscle-spasm and pain. Treatment: fixation 9 months, and then home without apparatus.

Nov. 7, 1926 (*Fig. 77*).—Perfect movements both hips.



FIG. 78.

CASE 6.—*Male. Age, 18 years.*

First seen March, 1916; had had intermittent pain and limp for 18 months.

Fig. 78.—Shows condition 11½ years after first examination. Was treated nearly 2 years by fixation, and for 6 months with no weight-bearing. Some limitation of abduction and slight limp, but patient can walk twenty miles.

CASE 7.—*Male. Age at onset, 4 years 4 months.*



FIG. 79.

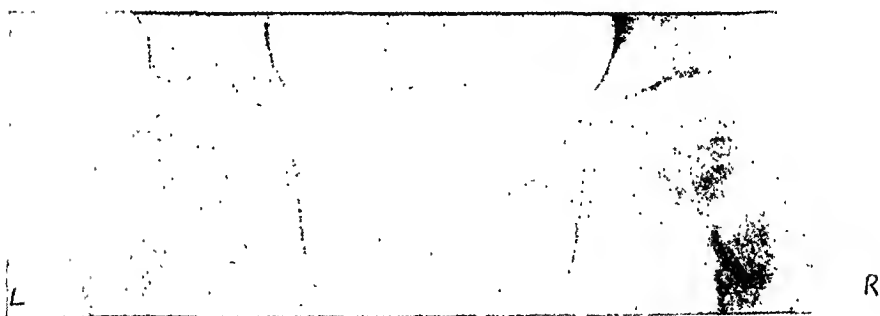


FIG. 80.

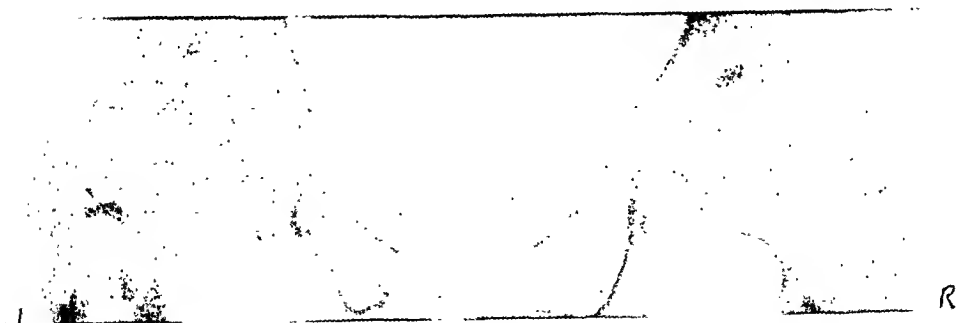


FIG. 81.

Feb. 8, 1923 (Fig. 79).—Taken at time of patient's first appearance. He came because of pain in the *right* hip, with considerable muscle-spasm. The *left* hip disease was not associated with symptoms, although at an earlier and more active stage than the right radiographically. Note metaphysial rarefaction (left).

March, 1924 (Fig. 80).—Patient has had 1 year's fixation of both hips; now no symptoms. Epiphyses of both femora are beginning to re-form. Now no treatment.

Aug., 1927 (Fig. 81).—Three years and 5 months after all treatment has been discontinued, and 4 years and 6 months after first examination. Movements are only very slightly diminished; the left hip in the direction of internal rotation, and the right in abduction. Very slight limp and no pain.

CASE 8.—*Female. Age at onset, 5 years.*



FIG. 82.

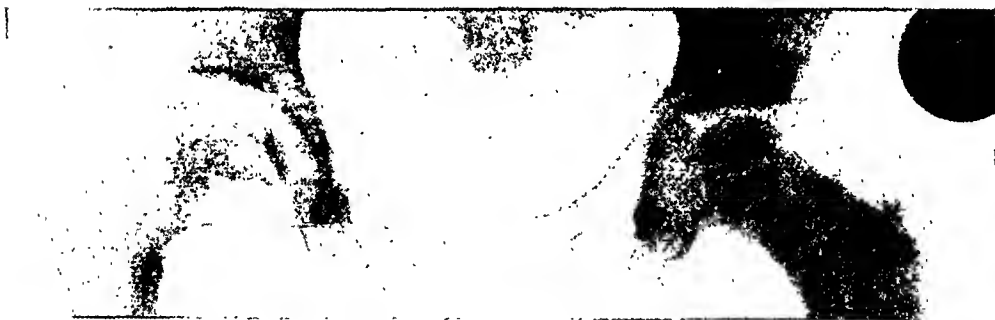


FIG. 83.

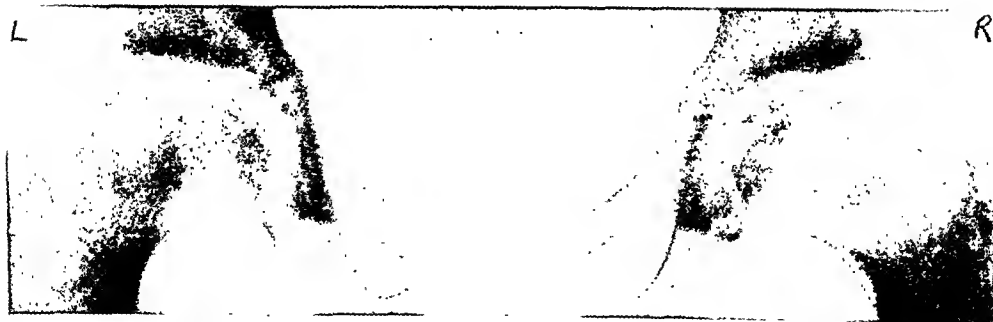


FIG. 84.

March 4, 1922 (Fig. 82).—First examination: slight occasional pain in left hip when tired, for nearly 1 year. Treatment: fixation 6 months, and then bed without apparatus 10 months. Note metaphysial rarefaction (left). Destruction of epiphysis has progressed.

July 5, 1923 (Fig. 83).—No symptoms in either hip. Sent home without apparatus. No limp and no pain.

June 8, 1925.—After 23 months at home, re-examined with history of slight pain in right hip for a week, with marked limp and limitation of movement. Treatment: 1 month fixation, and then discharged with slight limp and without apparatus.

Aug. 5, 1927 (Fig. 84).—Two years and 2 months after discharge. Left hip movements perfect. Right hip, very slight limitation of abduction and internal rotation.

CASE 9.—Male. Age at onset, 9 years 4 months.



FIG. 85.



FIG. 86.

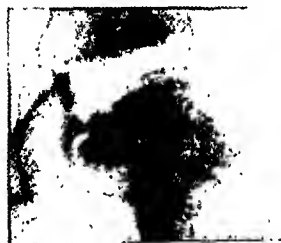


FIG. 87.

Jan., 1925 (Fig. 85).—History of 4 months' slight pain. Movements good. Treated for three months with extension, then no treatment.

Jan. 1, 1926 (Fig. 86).

Aug., 1927 (Fig. 87).—Two and a half years after first examination, and two years and four months after discharge. Practically no limp.

CASE 10.—Male. Age at onset, 5 years 3 months.

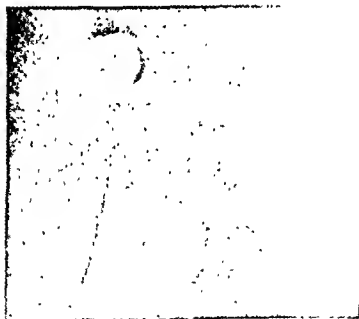


FIG. 88.



FIG. 89.

March 6, 1922 (Fig. 88).—History two months; only symptom slight limp. Note metaphysial rarefaction. Treatment: extension, four months; plaster, 12 months; walking with splint, crutches, and patten, 17 months.

Aug., 1927 (Fig. 89).—Three years and a half after treatment stopped. Movements perfect, no limp.

CASE 11.—*Female. Age at onset, 7 years 9 months.*



FIG. 90.



FIG. 91.

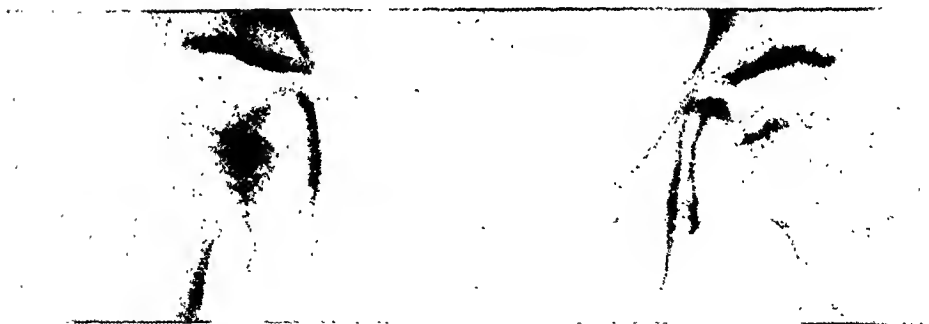


FIG. 92.

*Jan. 2, 1924 (Fig. 90).—*Limping one week, pain in left hip and knee, 1 in. wasting of thigh. Movements all good. No history of trauma. Admitted as tuberculosis and fixed for 9 months, then diagnosed as pseudocoxalgia; discharged with slight limitation of abduction only, and allowed to use limb.

*April 16, 1925 (Fig. 91).—*Six months after discharge, re-admitted with pain, left hip slightly flexed and adducted, movements all limited. Fixation for 8 months, and then no weight for a further 2 months. Note metaphysial rarefaction.

*Aug. 10, 1927 (Fig. 92).—*One year and a half after discharge. Slight abduction, slight limp when tired; otherwise no symptoms.

CASE 12.—Male. Age at onset: left hip, 5 years 5 months; right hip, not known.

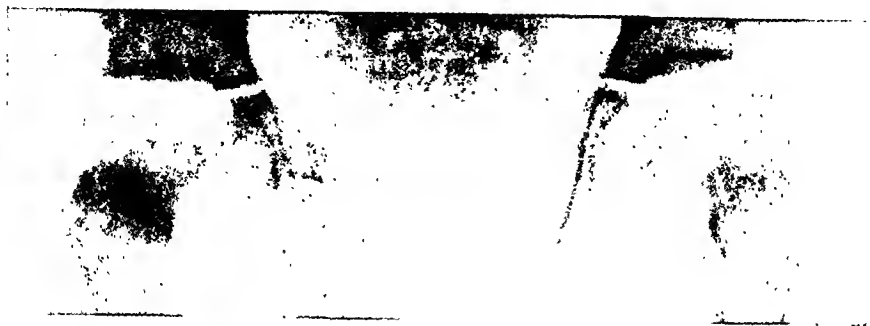


FIG. 93.



FIG. 94.

Feb., 1923 (Fig. 93).—For 5 months slight pain in left hip, no symptoms in right. On examination, marked pain and muscle spasm, with no movements. Fixed for eight months, then no treatment. Metaphysial rarefaction well shown in both.

Aug., 1927 (Fig. 94).—No treatment for 3 years 10 months. Movements perfect; very slight limp only after a long walk.

CASE 13.—Male. Age at onset, 5 years.



FIG. 95.



FIG. 96.

Jan. 4, 1925 (Fig. 95).—Slight pain and limp 1 year; now definite pain and spasm. Treatment: 3 months with fixation, then none.

Aug. 3, 1927 (Fig. 96).—After 2 years and 4 months without treatment. No signs or symptoms.

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CASE 14.—*Male. Age at onset, 4 years 8 months.*



FIG. 97.



FIG. 98.

July, 1925 (Fig. 97).—Six months' history. Very active disease; pain and spasm marked. Treated by fixation 4 months; all symptoms disappeared. Discharged without apparatus.
Aug., 1927 (Fig. 98).—After 20 months without treatment; abduction markedly limited, with a certain amount of resulting limp; otherwise no symptoms.

CASE 15.—*Female. Age at onset, 10 years.*



FIG. 99.

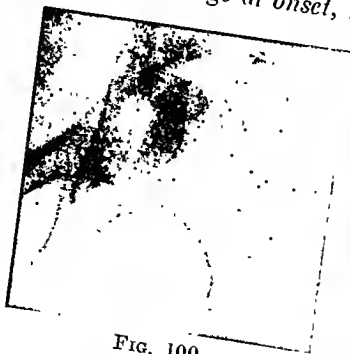


FIG. 100.



FIG. 101.

Aug., 1924 (Fig. 99).—History of slight pain occasionally, 1 year. Some flexion possible, but no other movements. Hip adducted and slightly flexed. Pain considerable.
Feb., 1925.—Disease progressing slowly in spite of treatment by fixation; no pain and limitation of abduction, and internal rotation only.

June, 1925 (Fig. 100).—A further stage of the disease. Treatment: fixation from admission for 2 years and 2 months, and then no treatment.

June, 1927 (Fig. 101).—Eight months after discharge. No symptoms; slight limitation of abduction.

CASE 16.

Oct., 1927 (Fig. 102).—End-result 6 years after onset. Limitation of internal rotation and abduction. Limp. Was treated for 9 months during active stage.

FIG. 102.

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SUMMARY OF 25 CASES, WITH SPECIAL REFERENCE TO THE END-RESULTS.

CASE	FIGURE NUMBERS	DEGREE OF ACTIVITY ON FIRST EXAMINATION	TREATMENT (In months)		END-RESULTS (Movements limited and limp)
			Fixed	Not fixed, but no weight-bearing	
1	62-64	Marked muscle - spasm. No pain	5	-	Perfect movements
2	65-70	Some spasm and pain ..	12	6	Internal rotation (slight)
3	71-73	Marked pain and spasm ..	9	4	Perfect movements, slight limp when tired
4	74, 75	<i>Left</i> : Marked pain and spasm	4	2	Internal rotation and abduction (marked)
		<i>Right</i> : No symptoms ..	6	-	Considerable limp
5	76, 77	<i>Left</i> : Very slight pain and spasm	9	-	Perfect movements
		<i>Right</i> : No symptoms ..	-	-	
6	78	Slight pain and spasm ..	20	6	Abduction, slight limp
7	79-81	<i>Left</i> : Pain and spasm ..	12	2	Internal rotation (slight)
		<i>Right</i> : No symptoms ..	-	14	Abduction (slight)
8	82-84	<i>Left</i> : Slight pain and spasm	3	3	Perfect movements, no limp
		<i>Right</i> : Pain and spasm very marked	1	6	Perfect movements, no limp
9	85-87	Some pain	3	-	Perfect movements, no limp
10	88, 89	No symptoms	16	7	Perfect movements, no limp
11	90-92	Pain and spasm (moderate) ..	12	6	Abduction, slight limp
12	93, 94	<i>Left</i> : Pain and very marked spasm	8	-	Very slight limp when tired
		<i>Right</i> : No symptoms ..	1	-	Perfect movements
13	95, 96	Pain and spasm moderate ..	3	2	Perfect movements, no limp
14	97, 98	Pain and spasm marked ..	4	2	Abduction very limited, limp
15	99-101	Pain and marked spasm ..	28	-	Perfect movements, no limp
16	102	Very slight pain and spasm ..	3	6	Abduction, internal rotation, slight limp
17	—	No symptoms	2	3	Perfect movements, no limp
18	—	Marked pain and spasm ..	2	-	Abduction, internal rotation, slight limp
19	—	Some spasm, no pain ..	6	4	Perfect movements, no limp
20	—	Pain and spasm very slight	8	4	Internal rotation (slight)
21	—	Some pain and spasm ..	10	2	Abduction and marked limp
22	—	Some spasm, no pain ..	8	-	Internal rotation, abduction, slight limp
23	—	Pain and spasm	30	-	Perfect movements, no limp
24	—	Pain and marked spasm ..	6	4	Internal rotation, and limp
25	—	Slight spasm, no pain ..	9	3	Perfect movements, no limp

CONCLUSIONS.

1. The majority of patients with pseudocoxalgia have some degree of pain and muscular spasm some time during the early stages of the disease.
2. The initial degree of activity of the disease does not necessarily bear any relation to the ultimate functional result.
3. The ultimate function of the affected limb is very good in the majority of cases. In patients in whom movement is limited the limitation is in the direction of abduction and internal rotation.
4. The length of treatment required to obtain a good result varies greatly, and does not depend on the initial amount of activity of the disease.

I desire to express my thanks to Dr. Veronica Dawkins for the preparation of the reproductions of the skiagrams in this paper.

REFERENCES.

-
- ¹ PERKINS, G., *Lancet*, 1925, ii, 295.
 - ² LEGG, A. T., *Jour. Bone and Joint Surg.*, 1927, Jan., 26.
 - ³ PERTHES, G., *Deut. Zeits. f. Chir.*, 1910, 107; and *Arch. f. klin. Chir.*, 1913, 101
 - ⁴ SUNDT, H., *Lancet*, 1921, i, 1153.
 - ⁵ PLATT, H., *Brit. Jour. Surg.*, 1922, Jan., 366.
 - ⁶ CALVÉ, J., *Rev. de Chir.*, 1910, July.
 - ⁷ GAUVAIN, H. J., *Lancet*, 1921, i, 1065.
 - ⁸ LEGG, A. T., *Surg. Gynecol. and Obst.*, 1916, March.
 - ⁹ NOBLE, T. P., *Jour. Bone and Joint Surg.*, 1925, Jan., 70.
 - ¹⁰ KIDNER, F. C., *Amer. Jour. Orthop. Surg.*, 1916, xiv, 339.
 - ¹¹ McWHORTER, G. L., *Surg. Gynecol. and Obst.*, 1924, May, 632.
 - ¹² PHEMSTER, D. B., *Arch. of Surg.*, 1921, March, 221.
 - ¹³ CALOT, F., *Presse méd.*, 1922, Jan. 14, 35.
 - ¹⁴ JANSEN, MÜRK, *Jour. Bone and Joint Surg.*, 1923, July, 528.
 - ¹⁵ BENTZON, P. G. K., *Brit. Jour. Radiol.*, 1926, Nov., 439.
 - ¹⁶ LEGG, A. T., *Boston Med. and Surg. Jour.*, 1910, Feb.

STAPHYLOCOCCAL SUPPURATIVE NEPHRITIS (CARBUNCLE OF THE KIDNEY).

By BRUCE M. DICK,

SURGICAL CLINICAL TUTOR IN THE ROYAL INFIRMARY, EDINBURGH.

THE term 'carbuncle of the kidney' has been in use for many years to describe an uncommon, but yet definite, type of suppuration of the kidney. The chief characters of the lesion denoted by this term are briefly as follows: There is an acute or subacute hæmatogenous infection of the parenchyma of one kidney by the *Sta. aureus*, the onset of which is frequently preceded by some trauma to the organ. The source of origin of the organism is most often a skin lesion such as a whitlow, boil, or carbuncle. The appearance of the kidney varies according to the stage at which the disease is encountered by the surgeon or pathologist; the most constant finding is that of multiple areas of necrosis or suppuration in the substance of the kidney, confined to one area of it. Fusion of the suppurative foci gives rise to a honeycomb-like necrotic cavity, or a circumscribed encapsulated abscess, and a localized enlargement of the affected area of the kidney. A perinephric abscess may result from the outward extension of the suppurative process, but eruption into the renal pelvis is unusual.

Review of the recorded cases of this condition indicates that it deserves to rank as a clean-cut pathological and clinical entity, and that its diagnosis may be arrived at with considerable accuracy. In one of the cases here recorded, recognition of the disease was facilitated by the pyelographic appearance presented by the kidney. The extent of the suppurative process and its relation to the structures of the kidney are demonstrated clearly by the accompanying coloured illustrations.

CASE RECORDS.

Case 1.—A young man, age 23, was admitted to hospital on account of pain in the right loin.

HISTORY.—A month before admission, while on a motor cycle, the patient had an attack of gnawing pain in the right loin which lasted for a few minutes. Two days later a similar attack was experienced, and he began to feel 'out of sorts'. From that time onwards he had repeated attacks of pain, which were at first slight, became more severe and continuous, and came on several times during the day, so that he was compelled to remain in bed. The patient was very feverish and uncomfortable, and on two or three occasions had rigors which were attended by nausea and vomiting. He got some ease by drawing the right thigh up. Shortly before admission to hospital, micturition had become more frequent, especially at night, but no alteration in the appearance of the urine had been observed. The patient

had lost his appetite, felt languid, and had become very thin; he sweated a great deal and felt his mouth always dry. He was very constipated. Two months before the onset of his illness a tooth had been extracted on account of an abscess at its root.

ON ADMISSION.—The man was very emaciated and looked ill. His face was pale and his lips dry and cracked. For comfort he lay with the right hip flexed. Temperature 99° , pulse-rate 92. Examination of the abdomen elicited the presence of a tender ill-defined swelling in the right loin which moved with respiration.

The patient was under observation for two weeks before an operation was performed. During this period the temperature fluctuated from 99° to 102° ; the pulse-rate remained about 90. The leucocyte-count was 12,000 per c.mm. Microscopic examination of the urine was carried out many times, but only on two occasions were pus-cells detected, and then only a few.

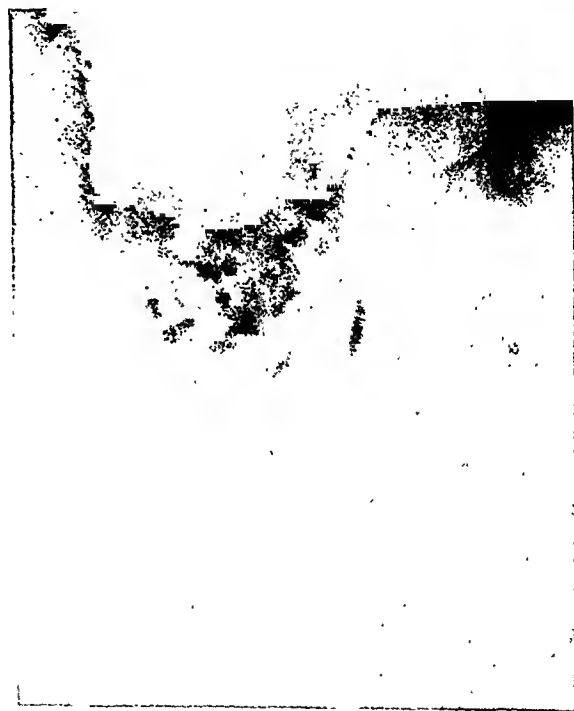


FIG. 103.—Case 1. Pyelogram of right kidney. Note the great reduction in volume of the renal pelvis and major calices. The minor calices are narrowed and deformed, and have assumed a 'frond-like' appearance.

UROLOGICAL AND RADIOGRAPHIC EXAMINATION. — On cystoscopic examination the bladder walls were seen to be healthy, and both ureters to secrete freely. Samples of urine collected from the right kidney, which was under suspicion, were sterile and contained no pus-cells. Each renal pelvis was injected with 14 c.c. sodium iodide, very slight discomfort being induced. The left pyelogram was normal, but the right showed a very striking alteration in form; the appearance is well demonstrated in *Fig. 103*, which shows that the lower major calix is very much narrowed, and that its minor calices have assumed a frond-like appearance. The upper calix showed a similar but less marked change.

OPERATION.—A perinephric abscess was found at the lateral border of the right kidney. About a teacupful of pus (which subsequent culture showed had been produced by *Sta. aureus*) was evacuated. The abscess communicated with the interior of the kidney by a small aperture on its lateral border; therefore the kidney was removed. Recovery was slow but satisfactory.

APPEARANCE OF THE KIDNEY.—The middle and lower part of the kidney were uniformly enlarged, and the capsule was a little thickened and adherent

over the swollen area. Below the centre of the kidney and at its lateral border there was a small ragged-edged opening from which purulent and necrotic material could be expressed. On section, the lower half of the cortex and medulla of the kidney were found to be the site of multiple areas of supuration, which in places were separate and sharply defined, but in other parts had become confluent and produced a spongy and irregular zone of necrosis. The lumen of the renal pelvis and calices was greatly narrowed (and in places constricted) by the adjacent inflammatory process. Penetra-

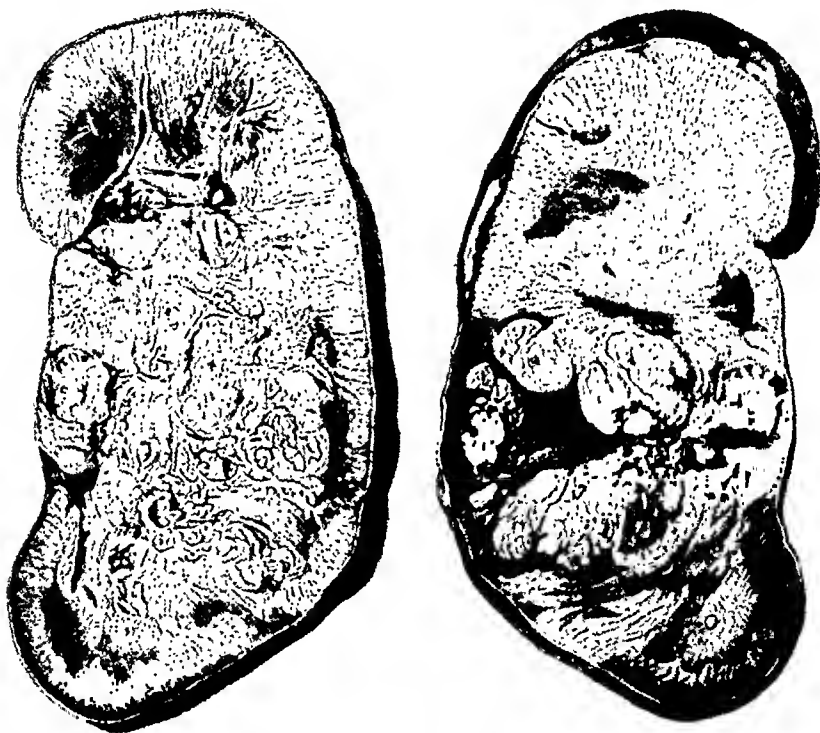


FIG. 104.—*Case 1.* Cut surface of kidney. The left figure shows an irregular necrotic mass in the kidney substance: calices are seen traversing the carbuncular area. The figure on the right shows multiple foci of necrosis, which, in places, are discrete and almost encapsulated: in other parts, suppurative areas have coalesced and broken down. Small concentric foci of necrosis are seen in the upper part of the kidney.

tion of the suppurative process into the calices could not be detected in large sections which were made of the whole kidney at different depths. Microscopic examination showed that the inflammatory foci were demarcated from the normal kidney tissue by a ring of newly-formed connective tissue. *Fig. 104* demonstrates the resemblance of this type of renal suppuration to that found in a carbuncle of the skin.

Case 2.—A well-developed youth, a miner, age 17, was admitted to hospital, under the care of the late Professor Alexis Thomson, for pain in the left loin.

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HISTORY.—For three weeks before he was sent into hospital he had felt weak and listless; the least exertion caused him to feel tired; and his appetite had become very poor. A week prior to coming to hospital, while at work, he was seized with acute pain below the ribs on the left side. The pain, which remained in the same position, got gradually worse, and he had to give up work and go to bed. Relief was obtained by a hypodermic injection of morphine. On the following day the pain was less severe, but he had a constant gnawing sensation in the left loin. Pressure or movement excited an attack of acute pain. He was most comfortable when he lay on his right side. The patient required to void his urine about ten times in twenty-four hours; his bowels were stubbornly constipated.

ON ADMISSION.—The patient was of good physique, but looked pale and ill. The tongue bore a thick brown fur, and the mouth and lips were dry. No septic lesion of the skin, mouth, or pharynx was discovered. Examination of the abdomen revealed very marked tenderness over the left kidney region and in the upper part of the left iliac fossa; tenderness on pressure was experienced also in the kidney angle behind. The left kidney could not be palpated. The temperature was 102.5° , and the pulse-rate 90.

The patient was under observation for nearly a month before an operation was performed; during this time the temperature varied from 99° to 102° , and the leucocyte-count ascended from 11,000 to 14,000 per c.mm. The urine contained neither pus, blood, nor organisms until a few days before operation, when a few leucocytes and red blood-corpuscles were found under the microscope. The discovery of these elements in the urine hastened the decision to explore the left kidney.

OPERATION.—The left kidney was explored by the usual oblique lumbar incision. The organ was found to be a little enlarged, and an ovoid swelling in the kidney substance was detected at its upper pole. The kidney was removed, and a rapid recovery ensued.

APPEARANCE OF THE KIDNEY ON SECTION.—An abscess, which was circumscribed by a thick ring of newly-formed fibrous tissue, occupied the upper and middle part of the kidney substance. The abscess extended outwards to within a quarter of an inch of the surface of the kidney. In the region of the hilum of the kidney the abscess was in contact with the outer wall of the upper major calix, which was narrowed and elongated (*Fig. 105*).



FIG. 105.—*Case 2.* Appearance of bisected kidney. The centre and upper part of the kidney contains a spherical mass of necrotic tissue. Note the encapsulation by newly-formed connective tissue, and the relation of the swelling to the upper calix of the kidney, which is narrowed and elongated.

Microscopic sections taken from the abscess wall showed a thick layer of newly-formed connective tissue that had enveloped the broken-down inflammatory products. Sections stained by Gram's method demonstrated abundant staphylococci in the interior of the abscess.

Case 3.—A married woman, age 48, came into hospital, under the care of Sir Harold Stiles, on account of pain in the right loin and severe debility.

HISTORY.—The patient had been in robust health until two months before her admission. Her complaint began fairly suddenly with a feeling of great tiredness which made her unfit for her household duties. She had severe frontal headaches, a 'chilly' feeling in the evenings, and great difficulty in getting her bowels to act. After a month she began to experience discomfort and constant aching in the right side below the costal margin in front. Attacks of severe stabbing pain, which did not radiate in any particular direction, began to be experienced; in the intervals, gnawing lumbar pain persisted. Pain was increased by any sudden movement or by deep inspiration, as, for example, a dry cough which supervened a week before she came to hospital. During this month she had frequency of micturition, especially at night. No change in the appearance of the urine had been noticed by herself or by her doctor.

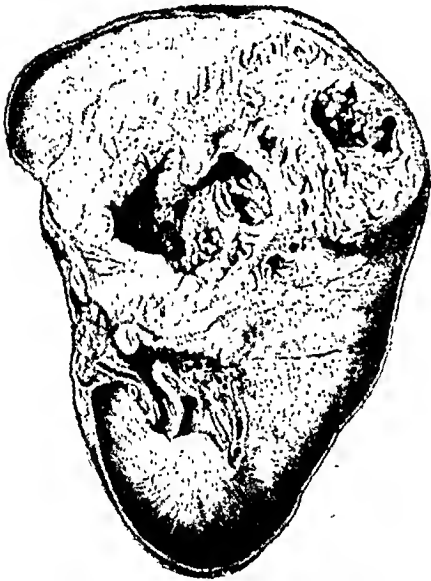


FIG. 106.—*Case 3.* Cut surface of left kidney. The upper part of the kidney is replaced by a cavernous necrotic mass. The remainder of the kidney is normal in appearance.

ON ADMISSION.—The patient was emaciated and looked ill; her complexion was muddy and her skin dry. The tongue bore a thick brown fur. Attempts to palpate the right kidney produced excessive tenderness both in front and behind. Pressure over the lower part of her thorax in front caused pain. The kidney could not be felt. On

auscultation, the breath-sounds were much fainter at the base of the right lung than the left. The temperature was 99°, and the pulse-rate 100.

The patient was kept under observation for ten days, during which period the pain was at times severe and she became more ill. The temperature was intermittent, with an evening rise to about 101°, and a morning fall to subnormal. Microscopic examination of the urine from time to time showed numerous pus-cells, and as a result of culture a growth of *Sta. aureus* was obtained.

OPERATION.—Although the pyuria was insufficient to cause naked-eye change in the urine, the case was considered to be one of pyonephrosis; operation was accordingly carried out, and the kidney removed. It was

enlarged and pyriform; the enlargement was confined to the upper pole, which felt soft and boggy, and a fluctuating swelling was palpable at its anterior surface. The capsule of the kidney was greatly thickened, and adhesions to neighbouring viscera made its removal a little difficult.

The patient made a satisfactory recovery except for the development of an apparently unrelated ischiorectal abscess which delayed convalescence for some time.

APPEARANCE OF THE KIDNEY ON SECTION.—The upper pole was replaced by multilocular abscess cavities which contained thick pus and necrotic material. The walls of the abscesses were lined by shreds of yellow sloughs. Areas of less advanced necrosis surrounded the main abscess cavities (*Fig. 106*). Intact but much narrowed and thickened calices traversed the diseased part of the kidney; the insertion of a fine probe along the lumen of the calices showed that their walls had resisted the invasion of the suppurative process.

The bisected kidney bore a striking resemblance to renal tuberculosis, and the true nature of the disease was only recognized after a pure culture of *Sta. aureus* had been grown from the contents of the abscess cavities. The staphylococci were stained *in situ* in sections taken from the diseased part of the kidney.

ETIOLOGY.

Some twenty-seven cases of carbuncle of the kidney are reported in the literature. The condition has received scanty recognition by British writers. The disease attacks men oftener than women; more frequent exposure to trauma is held responsible for its greater incidence in the male sex. Trauma, usually of the nature of a strain, is supposed to lower the resistance of the kidney and render it more vulnerable to organisms that, in a normal organ, would be eliminated without producing ill-effects (Thompson¹). This belief is supported by the experimental work of Brewer;² he found that the injection of bacteria into the circulation of 16 animals that had received trauma to one kidney resulted in the development of suppurative nephritis in the damaged kidney of 11 of the animals, and that the inflammatory changes began in the interstitial tissue of the kidney.

In carbuncle of the kidney the commonest portal of entry into the circulation for the *Sta. aureus* is a skin carbuncle, boils, or smaller multiple septic skin foci. Many other foci, such as a whitlow, a patch of eczema, and acute osteomyelitis, have been described. In *Case 1* of this report a 'root abscess' was considered to be the source of infection. Horn³ relates a case in which the disease followed an operation for removal of the prostate. The primary focus may be overlooked or not discovered, and it is of interest to note that symptoms of renal involvement may not appear until several weeks after the peripheral lesions have become quiescent, or have actually healed.

The earliest age at which the disease has been known to occur is 10 years (Barth,⁴ Colmers⁵), and the oldest 55 years (Reschle⁶). Only in one recorded case was the infection bilateral (Kretschmer⁷); in that patient, signs of involvement of the second kidney appeared ten days after nephrectomy had been performed on the other side.

CLINICAL FEATURES.

Symptoms come on gradually with malaise, headachic, and loss of appetite; the temperature is raised, and the patient may have repeated rigors, and he may vomit. Pain may be present from the outset, but often does not appear for a considerable time after the other symptoms (*Cases 2 and 3*). The pain is of a constant gnawing character, and is confined to the loin, the lower part of the chest, and the side of the upper abdomen; sharp exacerbations, excited by sudden movements or deep inspiration, occur from time to time. For comfort, the thigh on the affected side may be kept in a state of flexion. Urinary symptoms are generally absent; nocturnal frequency, and the passage of dark concentrated urine, may be noticed by the patient. As the disease progresses, the patient becomes extremely ill, sweats profusely, and becomes very emaciated. An irritating dry cough may develop. The bowels are very constipated.

The findings on physical examination will vary according to the duration of the illness. The abdominal muscles and the lower part of the thorax on the affected side show diminished respiratory movement, and a localized fullness or actual swelling may be seen in the loin. On palpation, defensive boarding of the lumbar and abdominal muscles is felt, and a very tender ill-defined mass may be detected in the lumbar region. The leucocyte-count ranges from 10,000 up to 25,000 per c.mm. Microscopic examination of the urine is most often negative, and catheter specimens are usually sterile; a few leucocytes and red blood-cells may be found from time to time, but are never abundant.

DIAGNOSIS.

A valuable aid in the diagnosis is the discovery that the patient has recently suffered from a septic skin lesion.

Pain in the lower part of the chest and fixation of the diaphragm on that side may lead to a suspicion of pleurisy or pneumonia; careful physical examination should be sufficient to exclude an intrathoracic lesion. An accurate history and careful abdominal examination should prevent confusion with retrocaecal appendicitis. In acute metastatic abscesses, in which the whole kidney is studded with small areas of suppuration, the urine usually contains a large quantity of pus and bacteria.

Pycnography should prove useful in determining whether a perinephric abscess has arisen from a renal carbuncle; discovery of a filling-defect in one or more calices would suggest an intrarenal origin of the abscess, and, in addition, would serve to decide the correct line of surgical treatment.

TREATMENT.

Nephrectomy is the most certain way of eradicating the disease, and when the operation is performed early the prognosis is good and recovery prompt. When the carbuncle is small, and restricted to one pole of the kidney, resection of the affected area has been recommended; cases will be few in which this method of treatment is practicable. Drainage of an actual abscess seen at operation has sometimes been successful.

In some of the reported cases a perinephric abscess had been drained and the renal origin of the infection overlooked or not suspected. In those cases, benefit was only temporary and fever soon returned; a secondary nephrectomy gave relief, and disclosed the origin of the perinephric abscess.

I should like to thank Mr. J. M. Graham for permission to publish the details of *Case 1*. I am obliged to Professor D. P. D. Wilkie for permission to make use of the specimen of the kidney of *Case 2*, and to Professor John Fraser for a similar privilege in *Case 3*. The urological investigation of *Case 1* was carried out in the Electrical Theatre of the Royal Infirmary, Edinburgh. The expense of production of the coloured illustrations was defrayed by a grant from the Medical Research Council, to whom I render my thanks.

REFERENCES.

- ¹ THOMPSON, T., *Lancet*, 1927, ii, 695.
- ² BREWER, G. E., *Jour. Amer. Med. Assoc.*, 1911, lvii, 179.
- ³ HORN, W., *Zeits. f. urol. Chir.*, 1924, xiv, 1.
- ⁴ BARTH, L., *Verhandl. d. deut. Ges. f. Chir.*, 1920, xlv, 94.
- ⁵ COLMERS, F., *Zeits. f. urol. Chir.*, 1924, xiv, 235.
- ⁶ RESCHLE, K., *Arch. f. klin. Chir.*, 1924, cxxix, 322.
- ⁷ KRETSCHMER, H. L., *Jour. of Urol.*, 1922, viii, 137.

OBSERVATIONS ON CALCIFICATION OF THE GALL-BLADDER: WITH THE PRESENTATION OF A CASE.

By J. J. ROBB,

LATE ASSISTANT TO THE PROFESSOR OF SURGERY, BRISTOL UNIVERSITY.

CALCIFICATION of the gall-bladder is a very rare condition, if we may judge from a search in the literature or in the museums. It seems worth while therefore to describe a new case of this condition and to attempt a classification of those specimens found in the Museums of the Royal College of Surgeons in London and Edinburgh.

Early in the perusal of the literature bearing on this subject it becomes evident that in fully 90 per cent of cases calcification of the gall-bladder is accompanied by gall-stones, and as the case to be recorded suitably exemplifies this, it will now be described.

CASE REPORT.

A. B. was admitted to the Bristol General Hospital under the care of Mr. Moore. The patient is the mother of eleven children. Prior to

the past eleven months she had been in perfect health, but since then her appetite has become poor. She has been subject to attacks of nausea without vomiting. In her right subcostal region she has had an almost continuous gnawing pain, also felt in her back around the medial angle of the scapula. This aching pain was increased by the ingestion of any food, fluid or solid. She has had no attack of jaundice.

ON EXAMINATION.—A well-nourished woman, looking in perfect health. Abdominal inspection reveals tenderness over the gall-bladder. Nothing is palpable.

OPERATION.—The gall-bladder was approached through a

right paramedian incision and found to be hard, and fixed by some periglandular adhesions. Its serous coat was thickened. Nothing abnormal

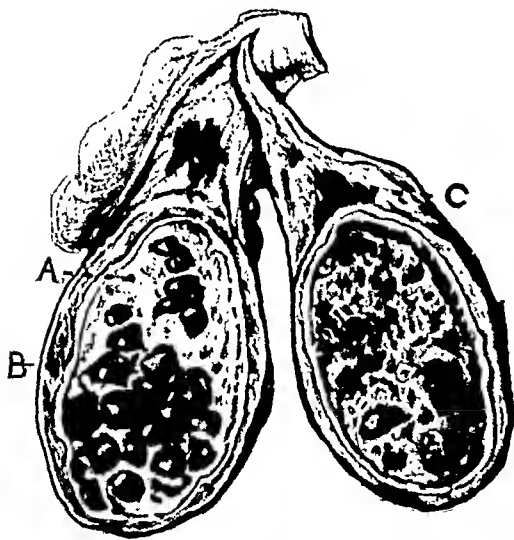


FIG. 107.—Calcareous gall-bladder. A, Calcium salts; B, Calcareous area; C, Cystic duct opened.

could be felt in the hepatic or common bile-ducts. The remaining structures in the abdomen were healthy. Cholecystectomy was performed. The patient made an uninterrupted recovery, and left hospital twenty-one days after her operation.

PATHOLOGICAL EXAMINATION (*Fig. 107*).—In shape the gall-bladder is that of a walnut, measuring from fundus to the entrance to the cystic duct 3.6 cm., and the greatest transverse diameter 2.6 cm. The serous coat is of a dull-grey colour, thickened and fibrotic. It can be stripped cleanly from the subjacent gall-bladder wall. Macroscopically the middle or muscular layer has the appearance of, and cuts like, bone. In thickness it averages 0.2 cm. The mucosa has entirely disappeared, and in its place is a layer of lime salts which completely encircles the wall of the bladder, crossing over and shutting off the entrance to the cystic duct inferiorly, but not extending into it. The gall-bladder is completely filled with laminated calculi, varying in size, the largest measuring 1.5 cm. across. These are rigidly cemented

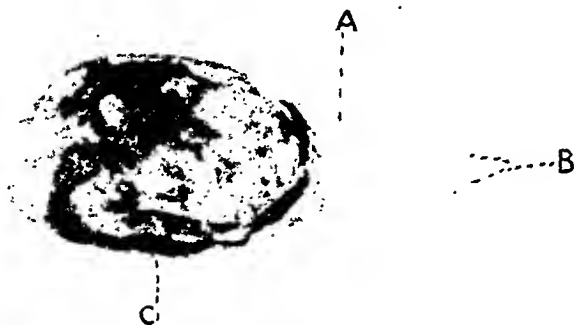


FIG. 108.—Radiogram of the gall-bladder taken after removal. A, Collum; B, Cystic duct C, Corpus.

together by the interposition of lime salts. The cystic duct shows no sign of calcification, nor does its lumen contain any calculi.

X-RAY EXAMINATION (*Fig. 108*).—A radiogram of the gall-bladder taken after removal shows the marked degree to which calcification has occurred, and the density of the calcareous matter deposited in the muscular layer of the wall. Again, it demonstrates the abrupt ending to the calcification immediately at the entrance to the cystic duct. X-ray photographs were also taken with a view to finding areas of sclerosis in other parts of the patient's body, but none were discovered.

MICROSCOPIC EXAMINATION.—A decalcified section from the gall-bladder wall was stained by hæmalum and cosin, iron hæmatoxylin and Van Gieson, Mallory's aniline blue and fuchsin. The mucosa is entirely absent. The muscular stratum has been wholly replaced by an acellular homogeneous substance—the decalcified layer. There is no evidence to suggest that this is of the nature of true bone, or even progressing towards bone formation. The inner and outer margins of this layer are well defined, and externally it is outlined by a thick coat of fibrous tissue.

OBSERVATIONS.

In this case the calcified gall-bladder is filled with calculi, these being cemented closely together with hard calcium salts, the whole surrounded by a thickened and fibrosed serous lining. The calcification of the wall is confined entirely to the parts surrounding the calculi, and does not extend beyond them in the direction of the cystic duct. This relationship is extremely significant, and is well substantiated by the case presented by Talbot. There the gall-bladder and the first part of the cystic duct both contained calculi and both were calcified, but the last inch of the cystic duct contained no calculi and was normal in appearance and size. It would seem therefore that a relationship exists between the calculi and the calcareous change in the adjacent wall.

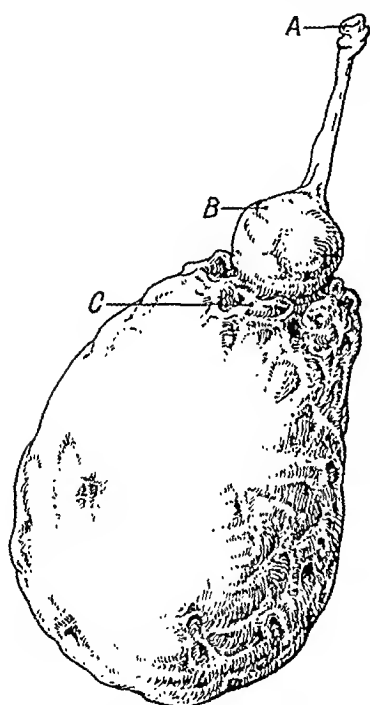


FIG. 109.—Drawing of Specimen 15(2)j2. A calcified gall-bladder with no calcification in the valvula spiralis. A, Cystic duct; B, Calculus impacted in cystic duct; C, Valvula spiralis.

ance and not a cholecystitis but a calcification results, where not infection but interference with function dominates the changing picture.

As a further and probably more convincing proof of these etiological factors, the specimen 641.1 O.C. 2808 A, presented by Dr. R. F. Barkwell, 1888, in the Hunterian Museum, London, may be cited. In it there is no evidence of any sort to point to an infective element having been present. The gall-bladder is completely calcified, and its interior is fully occupied by four cholesterol stones.

Realizing that the functional relationship is that of a contractile sac to an unyielding content, the sequence of pathological changes becomes apparent. The constant trauma of the muscular wall over the unyielding calculi is productive of degeneration and fibrous replacement of the essential tissues. The sac, because it is now merely fibrous tissue, contracts, gradually occluding its blood circulation, its means of vitality, slowly producing by a pressure atrophy a tissue such as in the body becomes calcified. Much emphasis has been placed in literature upon the infective element in etiology, but the insignificant part it plays in this process of calcification is reasonably obvious. In the example provided the calculi are of the mixed type, which may indicate that at some phase a degree of infection was present in the gall-bladder. At no time, however, was it sufficiently active to produce symptoms. Were bacterial infection at all a marked feature, such a stagnant, devitalized cyst, unable to evacuate its contents, would be extremely liable to become the nidus of an acute inflammatory process. Gall-stones with infective cholecystitis is the usual sequence. It is the rare exception in which the infection is nil or remains in abeyance.

Since it would seem to be true that by a contraction and pressure atrophy, calcification of the gall-bladder may result, it would also be reasonable to expect that a simple dilatation atrophy would result in a similar condition. Literature is void of help in this quest, but in the College of Surgeons' Museum, Edinburgh, a specimen of this condition is seen (*Fig. 109*).

Specimen 15 (2) j 2.—"From a woman, *æt.* 53, who died of carcinoma of the stomach. The gall-bladder projected about 7 cm. beyond the liver margin. The cystic duct was blocked by the only gall-stone which the gall-bladder contained. There was a history of a prolonged and severe attack of pain five years previously, but none of jaundice".

The sequence of changes ending in such a large, dilated, and calcified gall-bladder appears to call for little question: the lone cholesterol gall-stone impacted in the cystic duct with the resulting mucocele; the consequent distention and loss of function leading to atrophy in the muscular strata, fibrous replacement, and finally calcification. Again, it is noteworthy that the calcification has taken place throughout the entire corpus and surrounding the gall-stone. Beyond the impacted stone the cystic duct has remained normal in structure. The *valvula spiralis* (Heisteri) also, though intracystic, has maintained sufficient function to thwart degeneration and has remained uncalcified. It is therefore apparent that the stress of the dilatation is the chief causal factor in the degeneration, since only where stress has overcome function has calcification resulted.

Passing from these two types which we choose to term the non-infective types of calcified gall-bladder, we turn to the infective types, where an inflammatory fibrosis has terminated in calcification.

Osler, in his paragraph on calcification of the gall-bladder, remarks that "calcification may be a termination of suppurative cholecystitis." As a sequence of events it is a tempting one to accept. It provides for such a degree of inflammation as is bound to result in degeneration of the organ, and also a reason for the extreme rarity of the condition, for a true suppurative cholecystitis must as seldom spontaneously subside as calcification of the gall-bladder is rare. It may, however, be mentioned that, though it may be rash to preclude at this stage in our knowledge of body calcifications such a rapid and acute tissue necrosis as a possible precursor to calcification, nowhere in the histories of cases is there any evidence to suggest such a sequence of events. Rather are calcifying gall-bladders peculiarly void of symptoms, and particularly of acute symptoms, till their later stages are reached, when, ceasing to be a vital tissue, the body requires to deal with them. Sternberg writes: "As the result of long-continued chronic inflammation, the gall-bladder often becomes smaller, and can as a consequence of suppurative processes shrink into a scarcely cherry-stone-sized, dense, fibrous, not rarely also calcified body, enclosed in pseudo-membrane." Kaufmann states that as the result of chronic catarrhal inflammation, sometimes without stones, "the thick, calloused, often entirely smooth wall can calcify, even ossify." In these last two statements is found what appears to be the truth regarding calcification in association with infective conditions of the gall-bladder. It is the termination of a chronic cholecystitis. Nowhere in the history of the infection does an inflammation accrue sufficient in its acuteness

to produce a tissue destruction, but merely a tissue degeneration and fibrous metaplasia. The inevitable progressive contraction of scar tissue follows, with the slow elimination of its vitality and the deposition of calcium salts within it. Such a gall-bladder may or may not contain gall-stones. Though the calcification may involve both the corpus and cystic duct, the latter is always patent and not obstructed by a calculus.

An illustration of this condition is seen in the Hunterian Museum, London, which we reproduce in actual size (*Fig. 110*).

Thus far we have confined our remarks to that type of calcified gall-bladder classified by Osler as "the true infiltration of the wall with lime salts, the so-called ossification." There is, however, another type which does not mean a calcification of the whole wall of the gall-bladder, and which Osler also

mentions as "the incrustation of the mucosa with lime salts". An example of this condition is seen in the College of Surgeons' Museum, Edinburgh, 15 (2) j 3: "A gall-bladder distended and everted to show its coats, and on its inner surface a patchy deposition of lime salts". Here, though in a dried specimen, it is evident that this calcium deposit is merely in the mucosa, and that it may be shelled off in plaques, leaving the remainder of the wall of the gall-bladder intact. Fowler makes mention of yet another condition, "loose sand outlining the gall-bladder", an example of which is provided by Carman in the 1920 edition of his book. It would seem unnecessary to separate these two conditions. Both are depositions of calcium salts in the mucosa, and it is probable that the latter is merely an early stage in the development of the former. Etiologically we are dealing with a calcium deposition in these cases which cannot be explained as can the true infiltration of the



FIG. 110.—Specimen 2808. "A dried gall-bladder of very small size and having an extensive deposit of earthy matter like plates of bone in its coats. It is probable that previous to this deposit its coats were diseased. Presented by Sir William Blizard". The drawing represents in full size the bladder split longitudinally and opened out, so that the marked degree of contraction in its walls is apparent. No calculi were present.

wall, and neither histories nor specimens are available to assist us in elucidating the sequence of changes which lead to this selective calcification.

We would therefore classify calcified gall-bladders after the manner of Osler: diffuse calcification of the gall-bladder wall and selective calcification of the gall-bladder mucosa, the former being divisible into those resulting from non-infective atrophies and those resulting from infective atrophies.

The production of symptoms in these cases appears to be brought about in a variety of ways. The non-infective varieties usually produce no symptoms and are only discovered when the abdomen is explored for some other reason. Where symptoms do develop, they appear late in life and result from a periglandular reaction—an attempt to shut off from the body tissues, by a fibrous hyperplasia and adhesion formation, what has become simply a stone, calculi fixed in a calcium matrix set in a calcified body. Again, where the large stagnant cavity remains open to direct infection from its duct, it may become suddenly the site of an acute abscess without any previous

indication of gall-bladder inflammation being present. Such was the case in the example recorded by Haldane. In the chronic infective types the symptoms are those of chronic cholecystitis or cholelithiasis, extending over a period varying from fifteen to thirty years.

It is unnecessary to dwell on the mere numerical rarity in the incidence of calcification of the gall-bladder, a rarity which has been amply proved by Fowler in his many communications both with röntgenological and clinical authorities; but some comment may be made as to the reasons why this rate of incidence should be so low. Firstly, it is not the usual thing to find perverted or overstrained function alone producing atrophic changes in the gall-bladder. Secondly, it is unusual to find infection of such a sac as this remaining so indolent that over a prolonged number of years it may exist, yet be compatible with normal function. Lastly, the calcified gall-bladder is most often so quiescent in its development, so unproductive of symptoms, that the existence of many must never be known or even suspected, and they are cast upon the rubbish heap of treasures whose only signpost is senile decay.

I am indebted to Mr. C. A. Moore, Bristol General Hospital, for permission to utilize his case in this article, and to Professor E. W. Hey Groves for his assistance in the collection of material and details. To Sir Arthur Keith, Hunterian Museum, London, and to Mr. Greig, College of Surgeons' Museum, Edinburgh, who placed the specimens in those museums at my service, I also express gratitude.

BIBLIOGRAPHY.

- ALLISON, S. S., "Ossification of the Gall-bladder", *Lond. Med. Gaz.*, 1844-5, xxxv.
 BAYLESS, B. W., and SKINNER, C. (a case), *Internat. Jour. of Surg.*, 1925, xxx.
 BROOKS, S. T., "Ossific Degeneration of the Gall-bladder", *Trans. Vermont. Med. Soc.* 1890, liii, 55.
 CLAUDE, H., "Calcification de la Vésicule biliaire", *Bull. Soc. Anat. de Paris*, 1897, lxxii, 219.
 CARMAN, RUSSEL, D., *The Rontgen Diagnosis of Diseases of the Alimentary Canal*, 2nd ed., 1920. Philadelphia: W. B. Saunders Co.
 FOWLER, *Ann. of Surg.*, 1923, lxxviii, 623.
 GOTSTEIN, "Cholecystitis Calculosa", *Cor.-Blatt. d. Ver. dent. Aertz. in Reichenberg*, 1903, xvi, 2.
 GUTTMANN, P., *Deut. med. Woch.* 1890, xvi, 874.
 HALDANE, "Calcified Gall-bladder", *Edin. Med. Jour.*, 1874-5, xx, 836.
 LELOIR, H., "Calcification généralisée des Parois de la Vésicule biliaire", *Bull. Soc. Anat. de Paris*, 1881, vi, 444.
 OSLER, Sir WILLIAM, *Principles and Practice of Medicine*, 10th ed., 1925, 574. New York and London: Appleton & Co.
 PLUYETTE, "Vésicule biliaire ossifiée", *Red. d. actes du Comité méd. d. Bouches-du-Rhône*, Marseille, 1879-80.
 PHÉLIP and MAISONS, "Présentation d'une Vésicule biliaire complètement calcifiée", *Bull. et Mém. Soc. Anat. de Paris*, 1909, lxxxiv, 15.
 TALBOT, P., *Brit. Jour. Surg.*, 1920-1, viii.
 TOPINARD, P., *Bull. Soc. Anat. de Paris*, 1856, xxxi, 239.
 TUCK, "Complete Ossification of the Gall-bladder", *Boston Med. and Surg. Jour.* 1872-3, lxxxvi, 148.
 HUNTERIAN MUSEUM, LONDON.—Specimen 641-1, O.C. 2808 A. (Dr. R. F. Barkwell, 1888); Specimen 2808 (Sir William Blizard); Specimen 2806 (Dr. Goodhart, 1875); Specimen 2823 (E. Lloyd Bagshaw, Esq.); Specimen 2823 A (W. A. Carline, Esq., 1916).
 COLLEGE OF SURGEONS' MUSEUM, EDINBURGH.—Specimen 15 (2) j 2; Specimen 15 (2) j 3; Specimen 15 (2) j 4.

FRACTURES OF THE UPPER END OF THE FEMUR.

By J. J. M. SHAW,

ASSISTANT SURGEON IN THE ROYAL INFIRMARY, EDINBURGH.

"THE deep slumber of a decided opinion" is always a dangerous form of repose for the scientific mind, and a contribution which awakens new interest and stimulates informed discussion upon any subject becomes thereby a thing of value. A recent article in this JOURNAL¹ by George F. Stebbing is of this nature. It is original in

its treatment, and raises several fresh points for discussion in the subject of fractures of the upper end of the femur. Two challenging assertions are made: firstly, that violence applied in the region of the great trochanter with a breaking force produces as its usual result a fracture of the acetabulum; and, secondly, that the common means by which fractures of the intertrochanteric type are produced is by indirect violence due to sudden pressure by body weight upon the head of the femur with the limb fixed—or, we may assume conversely, by sudden violence exerted along the line of the shaft of the femur, the body weight fixing the head in the acetabulum.

The action to which Mr. Stebbing attributes the breaks through the trochanter (*Fig. 111*) would seem at first sight to be a reasonable result of the angulation of the bone at the junction of neck and shaft; a study of the divided bone and of skiagrams shows, however, that the main lines of support pass through from the shaft to the bearing-point on the head in a line, very slightly

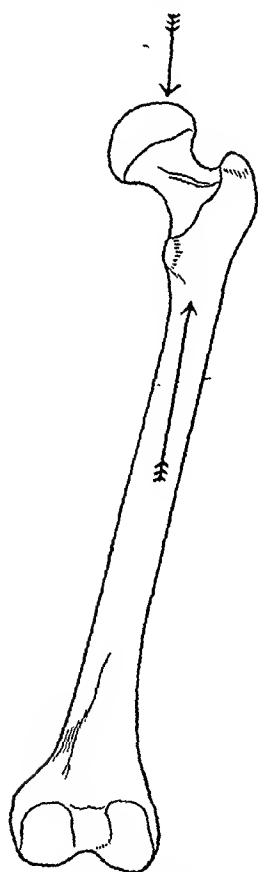


FIG. 111.—Application of forces to which fracture of intertrochanteric type is attributed by Mr. Stebbing.

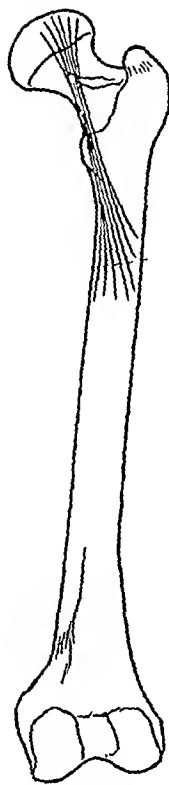


FIG. 112.—Lines of force in femur which indicate how intertrochanteric fracture is an improbable result of vertically applied forces.

curved, on the inner side. This pillar of compact bone normally carries the body weight, and it is evident that quite an exceptional degree of violence applied vertically would be necessary to break it (*Fig. 112*).

The usual effects of the descent of the body weight upon the femoral head with undue force are well seen in some cases of 'lift' accidents, where a rapid descent is suddenly checked, and the force is transmitted upwards



FIG. 113.—Comminuted fracture from a blow over the great trochanter in the cadaver.



FIG. 114.—Intertrochanteric fracture with comminution. Blow over trochanter.

through the lower limbs. The common injuries which are produced in this manner, or by other falls from a height in which landing is made on the feet with the knees extended, are fractures of the os calcis, of the upper end of the tibia, of the condyles or the shaft of the femur, and occasionally of the occipital bone. Fractures through the trochanter may in rare instances be produced in this way, and most text-books accept the possibility of an occasional fracture in this region by a fall upon the feet or upon the knees. The suggestion, however, that all fractures in the intertrochanteric region are due to this indirect violence, and never due to direct violence applied to the trochanter, runs strongly contrary to clinical experience.

In an attempt to verify these counter-assertions some simple experiments were carried out upon the cadaver, all the subjects having been over 65 years of age at time of death, and upon dried femora. In some fractures, and particularly in regard to displacement, it is legitimate to argue that the conditions obtaining in life cannot be faithfully reproduced after



FIG. 115.—Vertical fracture produced in a dry femur by vertically applied forces.

death. On the other hand, when a statement is made that a direct blow upon the trochanter never breaks the femur but causes fracture through the acetabulum, and one finds in the cadaver that a direct blow on the trochanter does fracture the femur and does not fracture the acetabulum, one must hesitate to accept the dogmatic statement. When the experimental evidence is supported by the weight of many generations of observers, the balance in favour of the old-time view remains exceedingly strong.

The experiments carried out were of the following nature. In the first cadaver a direct blow was struck upon the trochanter, the limbs being adducted



FIG. 116.—Multiple fractures of the pelvic girdle produced in the cadaver by slowly increased pressure over both trochanters. No acetabular injury.

and in slight external rotation. On one side a comminuted fracture of the trochanter occurred (*Fig. 113*), on the other a typical intertrochanteric fracture with comminution (*Fig. 114*). On the second body, in which, in the operative surgery class, bilateral excision of the knee had been performed, heavy blows were inflicted on the lower end of the shaft of the femur in all positions of the hip-joint. No effect was produced at the neck of the femur, or upon the acetabulum, and successive blows simply resulted in increasing local comminution at the point of impact of the mallet upon the lower end of the shaft. When the upper ends of the bones were exposed to be examined for damage, and when none was found, blows were directly applied to the

great trochanter, when comminution and fracture of the intertrochanteric type again resulted.

A dry femur was placed with the head on the ground and the shaft in vertical position. Blows were then applied to the condyles. In the absence of the shock-absorbing action of the acetabular bowl, slight flattening of the head took place, and finally an unusual fracture of the neck was produced in which the split ran approximately parallel to the pillar of compact bone (*Fig. 115*).

The third cadaver was placed in a large vice with parallel sides, and slowly increasing pressure applied over the two trochanters. A very great

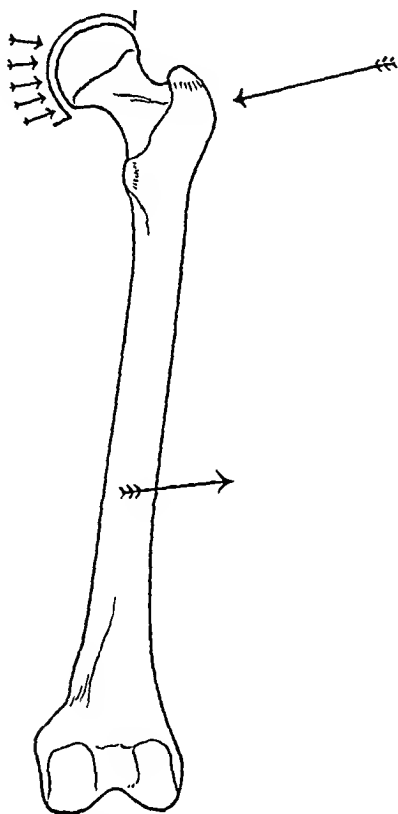


FIG. 117.—Leverage exerted by shaft as lower limb continues to fall when great trochanter is fixed against ground and head is fixed by body weight in acetabulum.

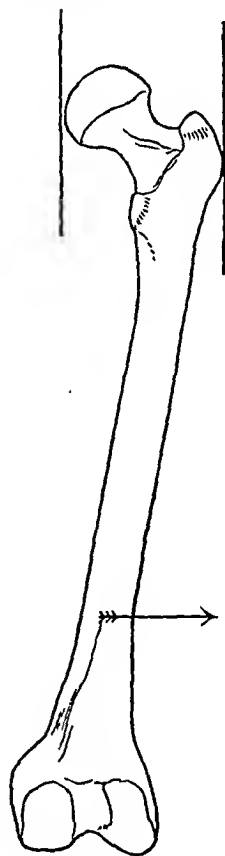


FIG. 117a.—Experimental reproduction of same.

degree of force was necessary before any sign of fracture occurred. Finally a definite crack was heard, and on dissecting away the soft parts it was found that fracture of the pelvis had taken place, as shown in the skiagram, in the horizontal pubic and the ischial rami (*Fig. 116*). Cracks were also produced in the ilia alongside the sacro-iliac synchondrosis. No trace of injury was present in the acetabula.

Another distribution of force was studied, which seems to be worthy of consideration. In the act of falling in a sideward direction the body tends

to sag at the pelvic level, and the trochanter and outstretched hand form the first points of contact with the ground. This is commonly seen in photographs of steeplechase and football falls, and is a well-remembered experience of those who have side-slipped on a frozen surface. In such a fall the upper end of the femur has for the moment two fixed points: the trochanter upon the ground, and the head of the femur fixed by body weight in the acetabulum. Powerful leverage is now exerted by the weight of the



FIG. 118.



FIG. 119.



FIG. 120.



FIGS. 121, 122, 123.

FIGS. 118-123.—Fractures through the base of the neck and the intertrochanteric region, produced by outward leverage of the shaft with the head and trochanter fixed.

still falling lower limb, with possibly the limb of the other side on the top of it, and this force, continuing to act, tends to break the femur at the trochanteric level. The type of leverage produced is shown in *Fig. 117*. If the upper end of a dry femur be held between two rigid flat surfaces which are in contact with the head and great trochanter, and leverage be applied by the shaft in an outward direction, exact reproduction of the common varieties of fractures through the trochanter follows (*Fig. 117a*). *Figs. 118-123* show a series of bones to which violence was applied in this manner. In only one of these is there a horizontal fracture.

The mechanism of the various types of fracture in dispute is probably as follows. Fractures through the trochanters, and through the basal part of the neck, are due to direct violence applied to the great trochanter, whether the action be a direct shattering one or an outward leverage when the trochanter is fixed against the ground. As far as experimental conditions can be allowed for demonstration of fracture, these two mechanisms have been effective.

It is highly probable that the rarer fracture of the acetabulum occurs only in those cases in which force is applied truly through the capito-collary axis (*Fig. 124*). This force is usually of the nature of a heavy fall in which the area of bone immediately below the great trochanter strikes a raised object such as the kerb. Some buffer-crushing injuries are also of this type. In such cases an impacted fracture of the neck into the shaft may occur, or the

acetabulum may give way. Where a slowly increasing force is applied over both trochanters fracture of the pelvic girdle is usually produced.

It has not been found possible in these few experiments to produce a fracture of the acetabulum by a direct blow, or by severe steady pressure over the great trochanter. It is conceded, however, that in very rare instances a fracture though the upper end of the femur which is atypical in line may be caused by the sudden descending weight of the body upon the head of the femur.

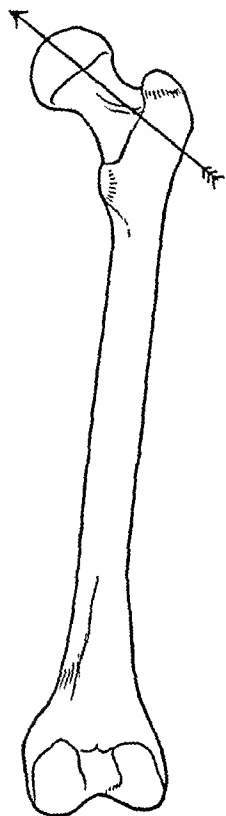


FIG. 124.—Force applied through the capito-collar axis likely to break the acetabulum.

SUMMARY.

1. Fractures of the narrow part of the neck are due to an axial twist of the femur, an indirect violence.
2. Fractures of the broad part of the neck, and intertrochanteric fractures, are due to direct violence over the trochanter, or to outward leverage of the shaft when head and trochanter are fixed.
3. Fractures of the acetabulum are due to force transmitted directly along the capito-collar axis, by violence applied immediately below the great trochanter.
4. Fractures of the pelvic girdle may be produced by the application of slowly increasing, powerful pressure over the trochanters.

These observations were carried out in the Surgical Research Department, University of Edinburgh. I am indebted to Professor D. P. D. Wilkie for permission to use the material, and to Mr. Frank Pettigrew, chief technical assistant, for the X-ray photographs.

REFERENCE.

- ¹ STEBBING, G. F., *Brit. Jour. Surg.*, 1927, xv, 201.

CLEFT PALATE.

(Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on Jan. 30, 1928)

By W. E. M. WARDILL,

HON. SURGEON TO THE TYNEMOUTH VICTORIA JUBILEE INFIRMARY.

IN the following paper a short account is given of cleft palate and its treatment. A new operative procedure, and its dangers and results to date, are described.

RESULTS OF OPERATION FOR CLEFT PALATE.

Of all the branches of surgery, that dealing with cleft palate is one of the most friendless. This I believe to be due to two very good reasons. Firstly, prolonged practice is necessary to enable the surgeon to attain sufficient skill in the type of operation chosen to assure a high percentage of successes in the closure of the cleft. Secondly, the results of any of the usually performed operations show such a very small percentage of good or perfect speakers that the surgeon requires an overdose of optimism in order to enable him to carry on.

From the standpoint of success in the closure of the cleft, the results are very encouraging. It is hardly fair, however, to expect the patient to show the same enthusiasm for a result in which the truly æsthetic value is more obvious to the surgeon than to himself. It is but cold comfort for him to possess a restored hard and soft palate and yet remain incapable of normal speech. Even so, he is almost always obliged to wear a dental appliance to supplement irregular or missing teeth; in which case why bother to repair the palate when practically the same effects are attainable by a skilful dentist? If the operation of gastro-enterostomy had to rely for its popularity upon such slender results as are obtained in cleft palate, the patients who would submit to such a procedure would be very few and far between. The present-day operations have, with certain modifications in technique, but not in principle, been practised for over a hundred years, and the time is now ripe to review the whole position and to draw up a profit and loss account. In this account the greatest item on the credit side must undoubtedly be the speech results. Union by first intention and considerations largely æsthetic should be given a very low value.

The aims in the operation of cleft palate are twofold: (1) To form a division between nasal and oral cavities whereby the patient is prevented from regurgitating food into the nose. The psychological effect plays a large part in the success of this. (2) To restore speech to normal, or, in the case of an infant, to enable it to speak properly in later life.

The first aim is achieved with a high percentage of successes in the hands of those skilled in this particular branch of surgery. There is no need to outline their methods, because they are well known and each practises

individual modifications, and on the whole the successes are of a fairly uniform high standard.

With regard to the second aim, the restoration of speech, the results are also fairly uniform; but in this case they are uniformly poor. By that statement is meant the dreadful fact that, out of all the operations done for cleft palate, there are very few who ever have normal speech restored. Usually it is found, where a patient speaks well after operation, that he was also a good speaker beforehand. On the other hand, a patient who speaks badly before operation will usually also speak badly afterwards. A further striking fact is observed—that many sufferers from cleft palate in whom no operation has been done at all are surprisingly good speakers. I have in mind a professional man who wears an obturator for a completely failed repair which was attempted in early youth (*Fig. 125*). I have heard people express surprise when they were told of his having a cleft palate. Moreover, he can speak almost as well without his obturator as with it. I believe that in such cases the result is largely attained by training, very often self-training, and that those who speak well do so more by their having done this than by having any material anatomical advantage over others. The first essential in this training is fundamentally psychological, and, as in any other uphill fight, depends upon an impelling desire to overcome difficulties, stimulated either by ambition or a sense of shame. Where these impulses are absent, no training, operation, or other mechanical device will ever succeed. The second essential to good speech is physiological, and as far as possible anatomical, restoration of the palatal mechanism. We know sufficient of the results of the present-day operations for cleft palate to be able to say definitely that the second essential is rarely obtained. What, then, can surgery offer to the sufferer from cleft palate? The answer is about a ten to one chance against his ever becoming a normal speaker, and the doubtful advantage of being able to deliver nasal speech with an intact hard and soft palate. The dental surgeon can do as much as this.

The object of this discussion, however, is not to deery the results of others but to see how they can be improved. Before tackling the problem, it is essential to have a clear idea of the anatomy of speech. Only that part directly concerned with the palate is relevant to this paper, and it alone will be considered in detail.

ANATOMY OF SPEECH.

During normal speech the soft palate is kept constantly in motion, being alternately drawn towards, or away from, the posterior pharyngeal wall. It forms part of a complicated muscular valve whose function is occlusion of the nasopharyngeal isthmus and the separation of the nasal and oral cavities. A cold in the nose frequently causes involuntary complete closure of this valve, and its effects are too well known to need any amplification. In normal speech, complete occlusion is necessary to enable clear pronunciation of all consonants except 'm,' 'n,' and 'ng'. Such efficiency is not so essential for the vowels. As the greater part of our language is made up of consonants it therefore follows that complete occlusion (cold in the nose) is fraught with much less disastrous effects upon speech than inability to occlude (cleft

palate). In cleft-palate speech the distinctness or otherwise will depend upon the relative amounts of sound projected through the mouth and nose. The nasopharyngeal valve is extremely active and moves with mercurial swiftness, and in very rapid speech must open and close many times in a second. It consists of two main parts, a palatal and a pharyngeal. The palatal part possesses two groups of muscles with opposing functions. One group, comprising the abductors, consequently opening the nasopharyngeal valve, consists of the palatopharyngei, the palatoglossi, and the tensores of the palate. The second, or adductor, group, closing the nasopharyngeal valve, is composed entirely of the levatores of the palate. Their action can be most clearly seen in a normal palate where 'Ah' is pronounced with the mouth wide open, a deep dimple forming on each side of the soft palate at the points of insertion of the muscles.

The pharyngeal part consists of an extremely complex muscle, the ultimate ramifications of which have not yet, to my knowledge, been adequately described. For this purpose it is sufficient to isolate those parts which have some practical bearing. The chief amongst these is the superior constrictor of the pharynx. In order to understand more fully the importance of this muscle it is necessary to diverge at this point. On examination of almost any unoperated case of cleft palate during the pronunciation of 'Ah' with the mouth wide open, a prominent ridge is seen running transversely across the posterior pharyngeal wall, to disappear into the upper reaches of the soft palate (*Fig. 125*). Passavant¹ was the first to describe this. If the bifid uvula be held aside during this movement, the outer end of the ridge is seen to enter the palate laterally to the muscular belly at the insertion of the levator on that side. The muscle appears to form part of the soft palate and, in contracting, it might be expected to exert some traction on this structure. This view of the structure of the muscle would appear to be morphologically compatible with what we know of its function as part of the nasopharyngeal valve or sphincter. Dissections of the pharynx of man and animals have shown this to be correct. With the help of my colleague, Mr. James Whillis, the superior constrictor has been shown to arise, not only from the usually described situations, but also from the palatal aponeurosis; a small fasciculus emanates from this situation to blend with the main portion of the muscle posteriorly (*Fig. 126*).

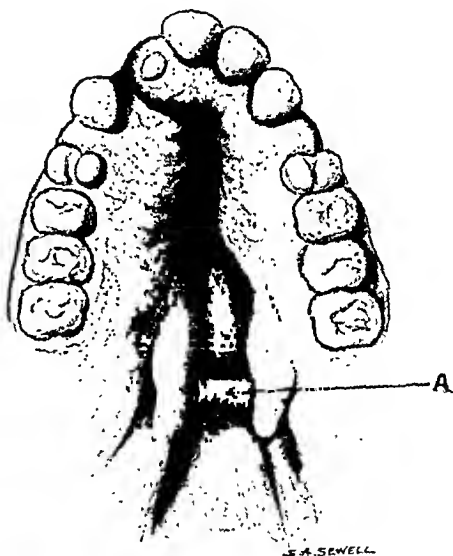


FIG. 125.—Cleft palate showing the ridge of Passavant (A).

In the resting state, the posterior part of the soft palate hangs down as a veil (*Figs. 126, 127*), parallel to, and not far removed from, the posterior pharyngeal wall. Between the two there is a narrow chink which is wide

from side to side, the cross-section area reaching a fairly large total. During speech this space is alternately obliterated and opened as mentioned before, but the muscles which take part in the movement make relatively small excursions, the width of the valve from side to side meaning that a slight

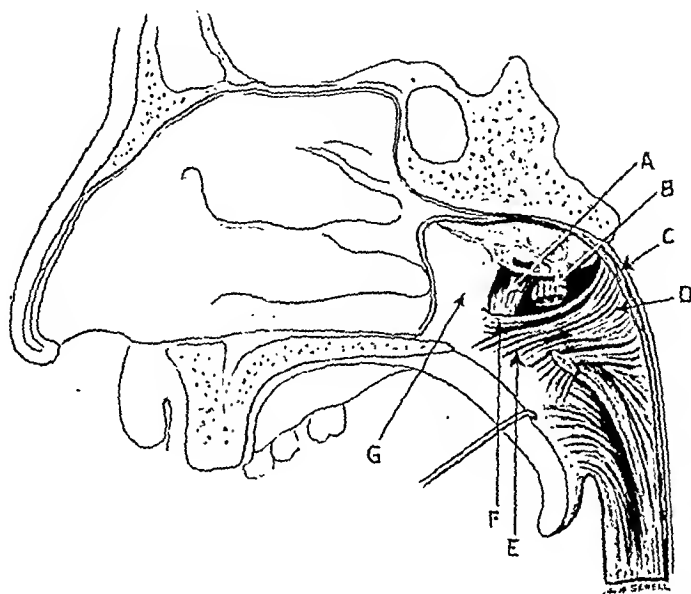


FIG. 126.—Dissection of normal nasopharynx. A, Tensor palati; B, Levator palati; C, Buccopharyngeal fascia; D, Superior constrictor; E, Fasciculus passing to soft palate; F, Fasciculus passing to internal pterygoid plate. G, Internal pterygoid plate.

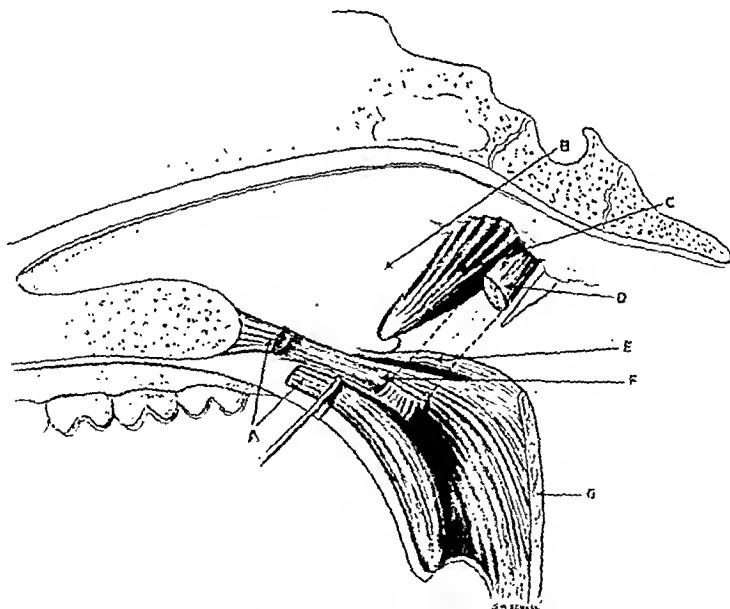


FIG. 127.—Dissection of nasopharynx of cow. A, Musculus uvulae; B, Internal pterygoid plate; C, Tensor palati; D, Levator palati; E, Fasciculus passing to internal pterygoid plate; F, Fasciculus passing to soft palate; G, Superior constrictor.

alteration in the antero-posterior diameter makes an immense difference in the total cross-section area. This principle is applied in the case of the valves of the petrol engine, where a small linear movement of the valve allows of a large cross-section area for the inlet of petrol vapour or the outlet of exhaust gases. Roughly, the average maximum antero-posterior excursion of which the soft palate is capable is about 15 mm. (Fig. 128).

The way in which this valve is operated is as follows. The superior constrictor muscle contracts and elevates the ridge of Passavant, thereby narrowing the nasopharynx antero-posteriorly and also from side to side. It remains in this state of contraction throughout speech, and only when speech ceases does it relax. The feeling of slow relaxation which normally occurs in the posterior pharyngeal wall on cessation of speech is readily recognizable by all who are conscious of their own muscular movements. Contraction of the superior constrictor and the formation of its transverse ridge are, then, probably for the purpose of forming a buffer against which the highly mobile

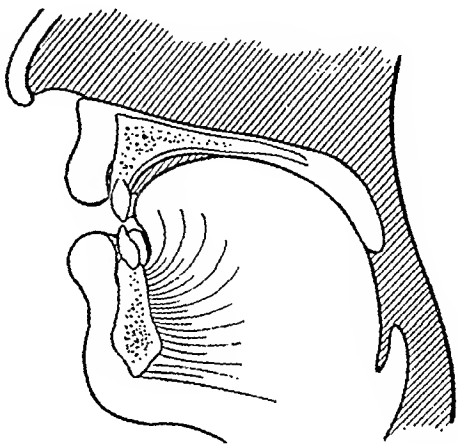


FIG. 128.—Tracing from skiagram. Normal palate: resting position.

soft palate can flap. The site of contact between palate and pharynx is at the level of the anterior arch of the atlas (Fig. 129). Once again, using the petrol engine as an example, the superior constrictor forms a valve seating for the upper surface of the soft palate. In order to appreciate to the full the principles of cleft-palate speech, great stress must be laid on the delicacy of the nasopharyngeal valve. As has been shown, the soft palate in the resting state is only about 15 mm. from the posterior pharyngeal wall, and its maximum linear movement cannot be greater than this. The width from side to side of the nasopharynx being considerably greater than this, it means that if the palate is unable to meet the posterior pharyngeal wall by so small a distance as 1 mm., a considerable cross-section area of pharyngeal airway is left open. The extraordinary delicacy required in this valve

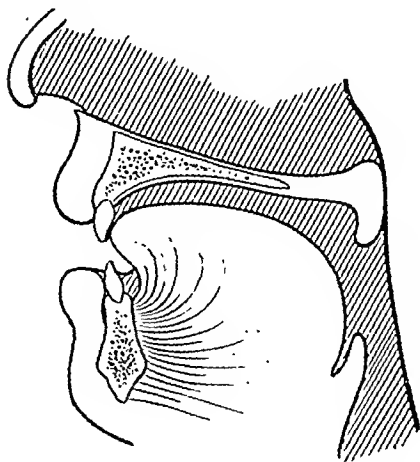


FIG. 129.—Tracing from skiagram. Normal palate: nasopharyngeal sphincter closed. This picture was made with the subject in the recumbent position, the nose being filled with bismuth emulsion. Note contact between soft palate and posterior pharyngeal wall.

should be properly appreciated. Moreover, it shows that examination of the palate by looking into the mouth is futile, because not even the keenest eye

could possibly appreciate a deficiency of a few millimetres between the palate and its valvular seating, when the essential parts are hidden from view. Most surgeons agree that many cases in which they have obtained apparently perfect anatomical results speak very badly, whilst others in which the anatomical result is apparently poor speak very well indeed. Let me repeat that the appearance of the anatomical results as seen from the mouth is no criterion of what is happening behind the palate, and as an observation is useless.

By the employment of an apparatus which I devised and demonstrated to the Royal Society of Medicine in June last, it was shown that normal speech depended upon a functionally competent nasopharyngeal valve, and that the worse the speech, after operation, the greater the deficiency in this respect. Further, it was shown in one particular case that noticeable cleft-palate speech persisted even when the total cross-section area of the deficiency of closure was as small as 48 sq. mm. Supposing the nasopharynx to be only 24 mm. in its transverse diameter (a gross underestimation of its size), this means that the palate in this particular case fails to meet the posterior pharyngeal wall by an average of 2 mm. The small size of the defect necessary to maintain noticeable cleft-palate speech is certainly very striking, and gives some indication of the extraordinary precision of the nasopharyngeal valve.

THE CAUSES OF CLEFT-PALATE SPEECH.

Cleft-palate speech results from two main causes: (1) An incompetent nasopharyngeal valve; (2) Incorrect speech habits designed to disguise this incompetence. I have proved this to my satisfaction by learning to mimic cleft-palate speech. That incompetence of the nasopharyngeal valve is not the sole cause is seen when the palate is paralysed as in diphtheria, the speech in this case being of an altogether different character from that of cleft palate. The difference lies in the fact that the subject of diphtheria has learnt normal speech; the tongue and lips go to their correct positions automatically; consequently the speech differs essentially from that of the cleft-palate speaker who has, in the course of his attempts to make conversation, developed incorrect anatomical habits. It is worth while at this point to examine some of these incorrect habits and see how the cleft-palate speaker, very often in spite of gross anatomical deformity, has been able to make himself understood.

Many cleft-palate speakers are able to say the letter 'k' with great facility. In the normal speaker this is done as shown in *Fig. 130*, where the pharyngeal valve is closed and the back of the tongue is forced against the roof of the mouth. Air is compressed behind the tongue, and its sudden release causes the explosive effort resulting in the sound 'k'. The cleft-palate speaker does this by throwing the tongue backwards and upwards towards the pharyngeal ridge of Passavant, and by completely blocking the cleft as shown in *Fig. 131*. Air is partially compressed in the space behind the tongue in sufficient quantity to make an explosive sound on release of the tongue. As this sound is not 'voiced', the quantity of air required is very small in amount. In the same way, many patients in the pronunciation

of the letter 's' learn to make the whistling sound, not between the teeth, but at the back of the pharynx between the tongue and the posterior pharyngeal wall.

The importance of understanding these abnormal tricks of speech is seen when one attempts to train a patient after operation. The letter 'k' has always previously been formed partly by a sort of cough and partly by releasing the tongue from its position in the cleft. After operation the patient is expected to close the nasopharyngeal valve, a function of which he has no previous experience, and while still keeping the valve closed to force air between the soft palate and the dorsum of the tongue. Acquisition of this simple feat is on a parallel with a man suddenly growing wings and being asked to fly. It can only be mastered by the practice of repeated trial and

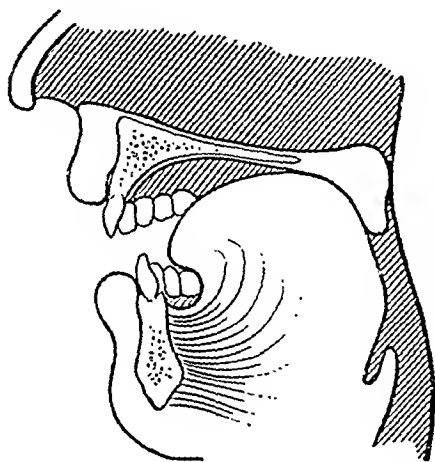


FIG. 130.—Tracing from skiagram. Normal palate during articulation of 'K.' Note contact between soft palate and posterior pharyngeal wall.

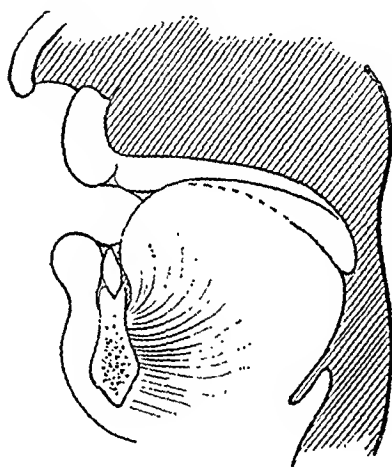


FIG. 131.—Tracing from skiagram. Un-repaired complete cleft of the palate during articulation of 'K.' No anatomical nasopharyngeal sphincter is seen.

error. In the same way the 's' has been made by whistling between the back of the tongue and the pharyngeal wall. This letter has to be shifted forwards to occupy the linguo-dental position. In some of my cases attempts have been made to study the best means of speech training after operation, and it is found that if the patient can once be made to produce a given sound by the proper method, repeated practice produces perfection. A useful way of making a patient acquire a letter, such as the 's', is to block up the nostrils with cotton-wool and then to demonstrate the correct positions for the tongue and teeth; a little practice soon brings the required result. The next step is to make the patient say the letter without the wool, closing, of course, the nasopharyngeal valve. The training of a cleft-palate speaker can only be likened to a normal speaker learning a new language, and in this connection one may be forgiven for quoting from the letters of Gertrude Bell, who, describing her efforts to learn Arabic, says "The pronunciation is past words, no western throat being constructed to form these extraordinary gutturals". If the patient, then, has acquired faulty methods of speech, he is faced with

the same difficulties as those mentioned by Miss Bell. Surely this is a great argument for performing any operation before speech has been acquired.

It is generally agreed that the results of the operation of median suture leave much to be desired, at any rate with regard to the all-important function of speech. The reasons for this are apparent in that this operation does not form a functional valve in the nasopharynx. It fails to do this because the soft palate is usually more rigid than normal, and is held in a position far removed from the pharyngeal wall. The two skiagrams shown (*Figs. 132, 133*) are from an adult of 45 years who recently underwent the *Langenbeck-Fergusson* type of operation. In the first the soft palate is shown in the resting position. In the second, the position of the soft palate is shown during the formation of the sound 'Ah'. It will be noted that the soft palate still remains at a considerable distance from the pharyngeal wall. This case is what is usually described as a very good anatomical result in that he

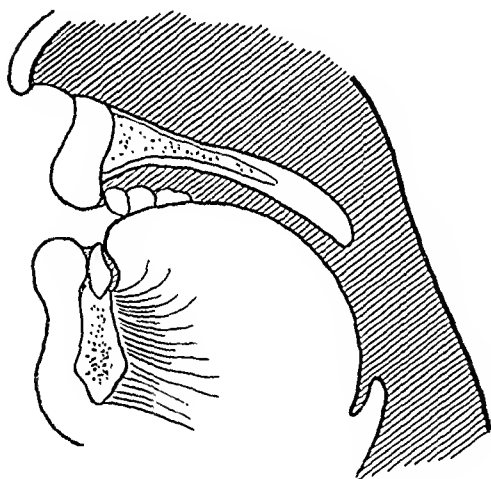


FIG. 132.—Tracing from skiagram. Case of cleft palate after *Langenbeck-Fergusson* operation; palate in resting position.

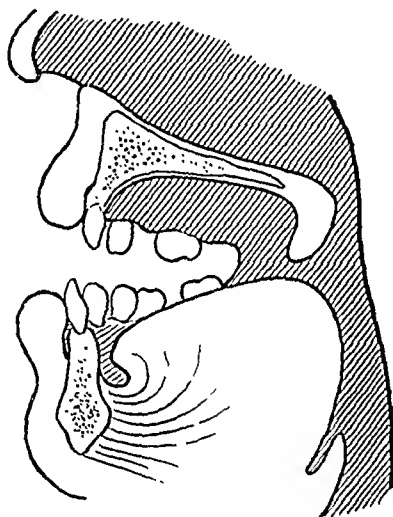


FIG. 133.—Tracing from skiagram, same case as *Fig. 132*, during pronunciation of 'Ah.' The soft palate, although mobile, does not reach the pharyngeal wall.

possesses perfect union and free movement of the palate. He is a very good speaker; but he was a very good speaker before operation and has really shown little more improvement than might be expected from the fitting of an obturator.

The speech tricks acquired by the subject with an open cleft have been mentioned, and the extreme value of the proximity of the tongue to the posterior pharyngeal wall has been shown. The way in which the tongue assists speech by blocking an open cleft has also been described. If, then, one substitutes for the cleft a repaired soft palate which cannot reach the pharyngeal wall, and also effectively prevents the tongue from so doing, speech will be worse, at any rate with regard to the posterior linguo-palatals. This is in accord with one's experience that very often, temporarily at any rate, speech is worse after operation.

ANATOMY OF THE DEFECT.

There are two great material changes in cleft palate: (1) *Defects in the soft parts*; (2) *Defects in the skull bones*.

1. **Defects in the Soft Parts.**—It seems to be generally agreed that the soft palate is shorter than in the normal, although this does not strike one in the accompanying drawing of a dissection of cleft palate (*Fig. 134*). One thing does, however, appear to be certain, namely, that the two halves of the cleft soft palate are not together equal to a whole normal palate. The musculature of the velum and nasopharynx shows no noticeable alteration from what can be seen in the dissections of the normal. On the other hand, in many cases

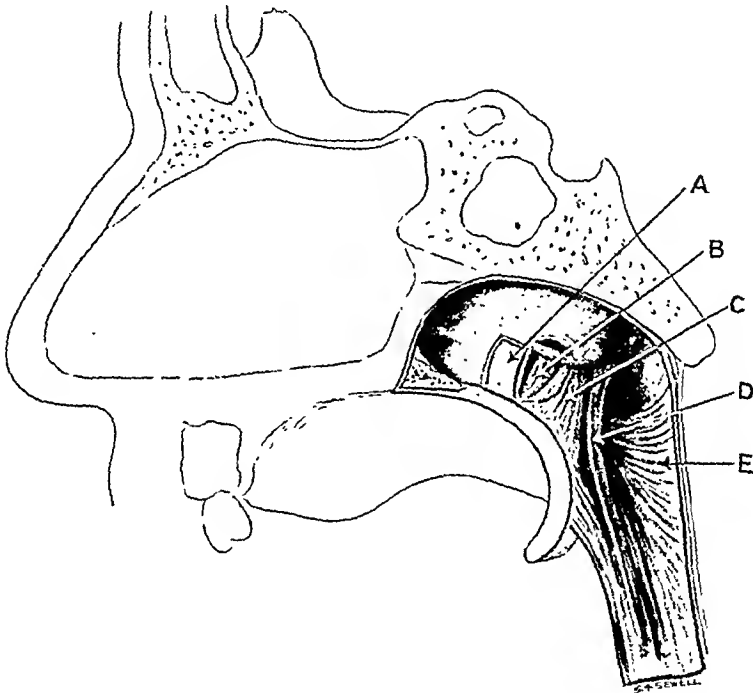


FIG. 134.—Dissection of nasopharynx in cleft palate. A, Internal pterygoid plate; B, Tensor palati; C, Levator palati; D, Salpingopharyngeus; E, Superior constrictor.

of unoperated clefts little or no movement can be seen in the muscles of the bifid palate or in the superior constrictor of the pharynx.

2. **Defects in the Skull Bones.**—Defects in the skull bones are more obvious than are those in the soft tissues. Firstly, in many cases of unilateral cleft, one finds gross asymmetry of the calvarium. The rough sketch shown in *Fig. 135* was made from a specimen of a complete left-sided cleft in the possession of Dr. Victor Veau, of Paris, who kindly placed his material at my disposal for examination. It will be noted that the skull is markedly asymmetrical, and that the left parietal eminence is displaced considerably further backwards than its opposite fellow. This is not merely a freak case, but can be seen in many specimens of unilateral complete clefts, and surely is an indication that the effects of cleft palate are not merely local, but are

associated with widespread structural changes in other parts of the skull and perhaps even further afield.

When normal skulls are compared with those possessing cleft palates, changes, which are amenable to measurement, are readily recognizable. Measurements have been taken for the purpose of proving the existence of

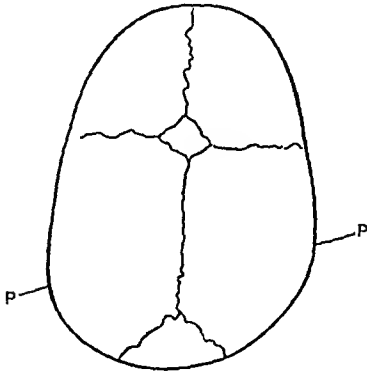


FIG. 135.—Diagram of skull bones from specimen of complete left-sided cleft, to show the backward displacement of the left parietal eminence (P). (Dr. Veau.)

bony defects in the nasopharynx. For comparison with normal skulls of about the same size, the distance between the glabella and occipital point, and the greatest transverse diameter, have been used in most cases; but in many the former distance has been replaced by that between the nasion and occipital point. This has been done because in many of the available specimens of cleft-palate skulls a large part of the calvarium had been removed.

CLEFT-PALATE SKULLS COMPARED WITH NORMAL.

		CLEFT PALATE (mm.)			NORMAL (mm.)		
		Nasion to Occipital Pt.	Trans. Diam.	Trans. Hamuli.	Nasion to Occipital Pt.	Trans. Diam.	Trans. Hamuli
Musée Dupuytren		180	153	38.5	176	148	35
"	"	162	149	43	171	133	28
Veau	23	156	133.5	32	160	133	33
"	2	127	117	27	151	124.5	23
"	0	123	103	24	148	124	26
"	51	122	101	24.5	133.5	117	23.5
"	—	121	102	31	120	92	21
"	10	117	111	31	112	90	18.5
"	—	101	82	23	107	84	22
"	12	100	94	26	103	90	19
"	192 F	93.5	88	24.5	99	82.5	19.5
"	—	93	83	25	96	82	17.5
		Glabella to Occipital Pt.	Trans. Diam.	Trans. Hamuli	Glabella to Occipital Pt.	Trans. Diam.	Trans. Hamuli
Nubian R.C.S.	..	175	128	41	178	149	34
Terat R.C.S.	..	187	150	42	190	147.5	36
D.U.C.M.	..	187	145	43.5	195.5	141.5	32.5
" "	..	169	125	39.5	180	141	30

The above measurements show that in cleft palate there is a definite bony increase in the transverse diameter of the nasopharynx. The increase is expressed only in terms of millimetres, and as such appears extraordinarily small in amount. But, as I have attempted to point out, the nasopharyngeal valve is of such precision and delicacy that even a minute change in its diameters will cause disastrous interference with its competence. How far these results are applicable to the living subject it is difficult to say. Moreover, the differences met with in cleft-palate subjects vary in the extreme.

One might expect that the patient with a cleft of the soft palate only would be easy to deal with and would give a good result. Experience shows that this is not so, the results even in this type of case being bad. I believe, but am unable to prove my belief, that there are bone changes in this type of case as in the others, and that very often the diameters of the nasopharynx in these cases are as much above normal as in many of the worse types of cleft, and that equally vigorous operative measures are as necessary in the one as in the other. The anatomical changes in the nasopharynx and palate are of the greatest importance in the consideration of repair. Firstly, we know that there is less than the normal amount of soft tissue, and that this has to be used to bridge an abnormally wide nasopharynx. Obviously, if the palate be sutured in the mid-line, its stretching from side to side will cause its shrinkage from before backwards. Secondly, each tendon of the tensor of the palate passes in a groove around the hamular process of the corresponding side to be inserted into the palatal aponeurosis. As the soft palate has been shortened by the mere process of suturing and stretching transversely, and as the widely separated hamular processes form the fixed bony points for the soft palate even after its separation from the hard, it follows that the palatal aponeurosis is bound to remain under tension, and consequently rigid, the upper surface of the soft palate being thereby prevented from reaching the pharyngeal wall. On theoretical grounds, suture of the soft palate without detachment from the hamular processes would seem to be one of the best ways of preventing the restoration of the nasopharyngeal valve. This, I believe, is one of the main reasons why the operations at present practised fail. At any rate, it probably accounts for one factor in failure, namely the immobility which often vitiates an otherwise apparently perfect repair.

THE SURGICAL PROBLEM.

The problem of the treatment of cleft palate is one in which surgery plays a very small but important part. Speech is essentially a material thing in that sounds are caused by vibrations in the air set up by vibrations in solid structures. In a musical instrument we can, by alterations in structure, vary the sound which will be emitted, but we do not expect to be able to produce musical sounds out of instruments which are structurally at fault. In the same way we cannot expect to restore normal speech to the cleft-palate sufferer unless he is supplied first with an instrument which is structurally and functionally sound. The greatest fault, common to all the usually practised operations, is the failure to restore a functional valve or sphincter. This, then, is the first fault to be remedied, and until this fault is rectified

perfect speech will never be attained. But, as has been shown, there is no operation at present in which a sphincter can be formed with certainty in every case. In fact, it is only in the most unusual cases that this happens, and with such infrequency that the result can only be regarded as fortuitous. If, then, a new pharyngeal valve is formed, the first step has been taken in the restoration of normal speech; but, even assuming the subject to be capable of using this valve, there are often other structural defects left which might detract to some extent from the quality of the speech emitted.

A NEW OPERATION.

The two-stage operation described below is based upon the preceding anatomical and physiological observations. It is founded upon the fact that the bony nasopharynx is increased in size, and that therefore any procedure which is content merely to restore a soft palate, however mobile, is bound to be a failure. Working on the principle that if the palate cannot be brought to the pharyngeal wall, the pharyngeal wall must be brought to the palate, the first stage of the operation was devised. The object of this stage is to narrow the nasopharynx laterally, and to produce a cushion of tissue on the posterior pharyngeal wall, imitating, but exaggerating greatly, that which is normally formed by the superior constrictor muscle, whereby a seating may be formed for the upper surface of the repaired soft palate. The operation is similar in principle to pyloroplasty, except that it is done the reverse way and for the purpose of producing the opposite effect.

First Stage.—The patient, lightly anæsthetized, is laid upon the back with a pillow beneath the shoulders and the head thrown well backwards. The mouth is held open with a gag. The pharynx is stimulated and the position of the ridge of Passavant noted. With a fine tenotome, the mucous membrane is incised transversely at the level of the ridge of Passavant, i.e., over the anterior arch of the atlas, and is then held open with fine sharp hooks (*Fig. 136*). Care is taken not to incise too deeply and thus open the loose areolar tissue between the buccopharyngeal and prevertebral layers of fascia. The buccopharyngeal fascia is relatively tough, and the superior constrictor muscle and its covering mucosa can be readily peeled or scraped from it by means of blunt dissection with a curved aneurysm needle. The dissection is carried on in this layer, upwards nearly as far as the base of the skull, downwards for a similar distance, and laterally a little beyond the ridge caused by the salpingopharyngeus muscle. During the whole of this part of the operation some resistance is felt to the dissection. If this resistance is not felt, it is probable that the point of the needle is in the loose retro-pharyngeal cellular tissue.

Using the aneurysm needle as a combined director and retractor, the incision is enlarged laterally right up to the salpingopharyngeus by the use of curved scissors (*Fig. 137*). Bleeding is very small in amount, there being little more than a slight ooze. The business of sewing up the incision in a vertical direction is now undertaken. I have had a small spring wire retractor made (*Fig. 138*) for the purpose of facilitating this procedure. The retractor holds the incision open in a vertical direction. Using very small curved

needles made on the Mayo principle, sutures of No. 0 catgut are inserted, commencing first at the upper and lower ends of the wound ; the sutures are left long and may be used as retractors. It is important to take big bites of tissue on each side, as the sutures have a tendency to work out very quickly. The suturing is completed in the centre of the wound by joining together the two salpingopharyngeal folds, including a sufficient bite to obtain a firm hold

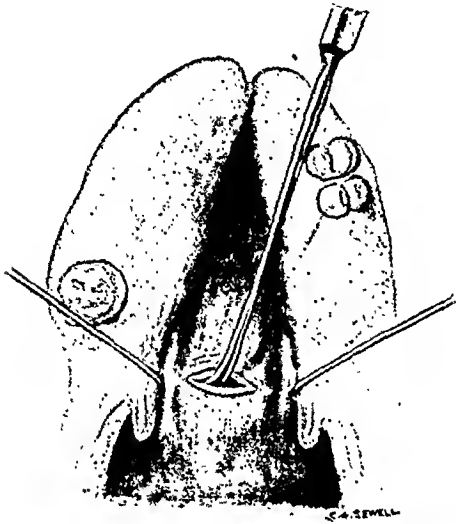


FIG. 136.—Separation of the superior constrictor from the buccopharyngeal fascia.

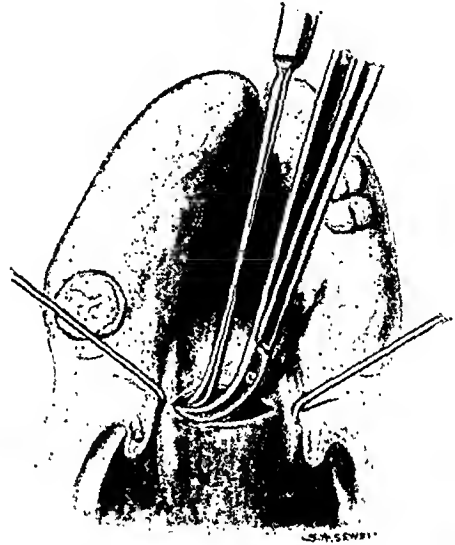


FIG. 137.—Extension of the incision as far as the salpingopharyngeus.

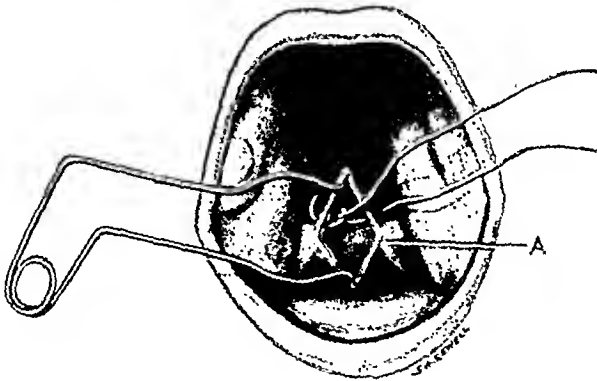


FIG. 138.—Suture of the wound in a vertical direction. A, Edge of salpingopharyngeal fold.

of the tough connective tissue which is intimately bound up with these. Usually a suture is passed through the posterior pillars of the fauces for the purpose of relieving the tension for a few days. At the end of the operation the halves of the soft palate are found to be much closer together, and the halves of the uvula may overlap. In fact one wonders how the patients are going to be able to swallow.

For the first few days the sutures hold and all appears to be going well.

There is little or no complaint of pain. About the fifth day the suture holding the salpingopharyngeal folds gives, and a small hole appears. Usually no further breaking-down occurs, and a permanent ridge on the posterior pharyngeal wall remains.

Second Stage.—The second stage of the operation is the repair of the palate, and is done after the wound of the first stage is healed and the tissues are once more in a settled state. This usually requires an interval of about three or four weeks. Either the Langenbeck-Fergusson or the Gillies-Fry method is suitable for this purpose, with the modification of division of the hamular processes or of the tendons of the *tensores* of the palate, thereby setting free the palatal aponeurosis, and so destroying the greatest bar to backward displacement of the palate. On theoretical grounds this step must be regarded as of the greatest importance, and in my experience it has certainly justified its performance. Whether in practice the simple Langenbeck-Fergusson or Gillies-Fry operation will suffice when combined with pharyngoplasty, remains to be seen. In seven cases I have done the Langenbeck-Fergusson, and in one the Gillies-Fry, operation. The large hole left between the hard and soft palates in the latter procedure offended my æsthetic sensibilities and drove me to do the Langenbeck-Fergusson as a routine.

Whatever the procedure adopted in the second stage, a few words of warning are necessary. If the first stage of pharyngoplasty has been successfully performed, repair of the palate completely blocks the nasopharynx for a matter of a few days until the sutures loosen. It is therefore extremely important that a careful watch be kept over the patient until recovery from the anæsthetic is complete. Ignorance of the possibility of this complication in one of my cases was nearly the cause of a fatality. Since then I have been in the habit of passing a silkworm-gut suture through the tongue for the purpose of traction. For the first few days breathing through the nose is often impossible, and, in order to accustom the patient to this before operation, in the last case of my series, the nostrils were blocked with cotton-wool for forty-eight hours beforehand.

Both stages of this operation can be done at one sitting, as in three of the cases recorded below. I do not think this is wise, because once the soft palate is repaired, the difficulties attendant upon any necessary subsequent interference in the nasopharynx are increased considerably. Moreover, in the two-stage operation one can study the effects of the first before proceeding to the second.

Dangers.—All operations, however trivial, have certain risks attached to them, and a certain mortality associated with these risks. The operative procedure which has been described is no exception to this rule. The dangers as known at present are mediastinitis, asphyxiation, and deafness.

Mediastinitis.—In my series of nine cases I have had one death which, in the light of further experience, was avoidable, and I am firmly of the opinion that similar occurrences will be few and far between. The fatal case referred to was that of a boy of 4 years, in whom was done the first stage or pharyngoplasty. He was the eighth of the series, and in the desire to obtain thorough separation of the superior constrictor muscle, it was not realized

that the buccopharyngeal fascia had been perforated, and that the dissection was being carried on in the loose retropharyngeal connective tissue. The operation was completed in the usual way. The child died thirty-six hours later from acetonaemia. At the autopsy a mild degree of retropharyngeal cellulitis was found, with a small purulent track passing down into the mediastinum. Although it is not thought that this child died from sepsis, it is preferable that the case should be labelled a direct operative death. If one is careful not to perforate the buccopharyngeal fascia, a repetition of this unfortunate complication should not occur. It is most strongly urged to anyone who might contemplate this operation to be most careful in the study of the anatomical facts, and, before attempting to perform it on the living body, to try it in the cadaver in order that a clear conception of the limits and texture of the buccopharyngeal fascia may be obtained. This is not an operation to be undertaken lightly, and before I myself first did it on the living subject, I had made many dissections of the cadaver.

Asphyxiation.—In discussing the treatment after the second stage, or repair of the palate, the danger of asphyxiation was mentioned. This is repeated here in order that it may be duly recorded on the debit side of the account.

Deafness.—Two of my patients have complained of deafness after the second stage of this operation. In one it was only transitory, in the other slight deafness still persists. Professor Grey Turner tells me that he has occasionally noticed the same complication after the Langenbeck-Fergusson operation.

Whether the dangers and disadvantages of the operative procedure recommended will in the end outweigh the advantages, time and experience alone will show. Full exposure of what are thought to be the disadvantages has been made not for the purpose of discouragement of others, but in order that others may help to put the operation in its proper place, either by its acceptance or rejection.

RESULTS OF THE OPERATION.

Case 1.—The first patient upon whom this new procedure was attempted was a minor, age 17 years. He had a cleft of the soft palate and about half of the hard. He had been operated on twice, at the ages of 3 years and 3½ years. Both operations were complete failures and the palate broke down. What was left of the soft palate was scarred and its mobility was to some extent interfered with.

FIRST OPERATION (Feb. 8, 1927).—The patient was anaesthetized and a transverse incision was made through the mucous membrane about the level of the ridge of Passavant. The superior constrictor muscle was separated from the buccopharyngeal fascia for a distance of about an inch, but the salpingopharyngeal folds were not interfered with. The wound was then united in a vertical direction. At the end of the operation there was a distinct elevation of the posterior pharyngeal wall, and the two parts of the uvula were found to have been drawn much closer together. The whole procedure occupied over an hour, difficulties being encountered owing mainly to unsuitability of instruments and clumsiness of myself. The patient made an uninterrupted recovery from this stage, and no complaint of pain was made. The wound healed very well but not by first intention, a small area being left to granulate. At the end of three weeks there remained a small elevation of the posterior pharyngeal wall.

SECOND OPERATION (March 19, 1927).—The soft palate was separated from the hard and the hamular processes were divided. Repair was done according to the

method advocated by Gillies and Fry. Recovery from the operation was uneventful and later the patient was fitted with an obturator.

The soft palate, as might be expected from the pre-operative condition, was not very mobile. There was no evidence that the pharyngeal sphincter was competent. Speech remained of the nasal type. There were no facilities for speech training, but the parents expressed the opinion that speech was much improved. Of this I am not greatly convinced.

Case 2.—S. C., age 24, miner. Wide congenital cleft of the soft palate and all the hard palate as far as, but not including, the alveolar border. Speech very bad. The ridge of Passavant was not well developed, and there was poor movement in the levatores of the palate.

FIRST OPERATION.—A small transverse incision was made through the pharyngeal mucosa. The superior constrictor muscle was separated and sutured in the vertical direction. No marked narrowing produced. The salpingopharyngeal folds were not sutured. After-progress was uneventful, and at the end of three weeks there was little to be seen of the original operation.

SECOND OPERATION (Aug. 19, 1927).—Repair of both hard and soft palates by Langenbeck-Fergusson method with division of the hamular processes. Short lateral incisions were extended around the last molar teeth. There was difficulty with regard to the suture of the hard palate, so the anterior extremity was left open. Recovery was uneventful except for the breaking-down of some of the hard-palate sutures. There was no evidence of a pharyngeal sphincter having been formed. Speech was improved, but not to any marked extent. Slight deafness was complained of shortly after the operation, and has persisted.

Case 3.—G. M., age 13. This girl had a cleft of about the posterior three-quarters of the soft palate. The nasopharynx appeared to be very large both anteroposteriorly and transversely. Speech was very bad indeed.

OPERATION (Sept. 30, 1927).—The mucosa of the pharynx was divided transversely and the superior constrictor separated, although not very freely. The wound was sutured vertically, with six stitches of catgut, the salpingopharyngeal being included. Small lateral incisions were then made and the hamular process on each side was divided. After paring the edges, the soft palate was united in the middle line. Recovery was uninterrupted.

Speech in this girl was much improved as a result of the operation. The nasopharyngeal valve was restored, as evidenced by her ability to snort—that is, she can vibrate the soft palate against the posterior pharyngeal wall during the act of drawing mucus from the back of the nose into the mouth.

Case 4.—Dora S., age 36. Complete cleft of hard and soft palates, including the left side of the alveolar margin. Hare-lip repaired in childhood. Ridge of Passavant not well-developed; movement of levatores not very good. Speech poor.

FIRST OPERATION (Aug. 31, 1927).—Formation of a ridge on the posterior pharyngeal wall by the usual method. No particular care was taken with the salpingopharyngeal folds. After the operation there was no complaint of pain and the wound healed in the usual manner. A well-marked ridge remained on the pharyngeal wall. The patient's friends noted an immediate improvement in speech, which was not, however, apparent to her medical attendants.

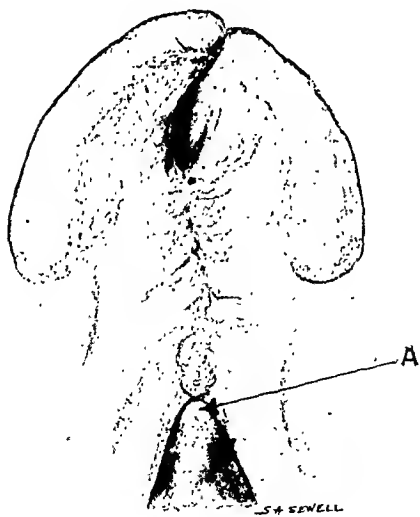


FIG. 139.—*Case 4.* Condition nine weeks after operation. A, Lower edge of pharyngeal ridge.

SECOND OPERATION (Oct. 7, 1927).—The hard and soft palates were repaired according to the Langenbeck-Fergusson method with the modification suggested.

Recovery was uninterrupted and the palate healed except for a small hole behind the alveolar margin (*Fig. 139*). The soft palate was fairly mobile. This patient possesses a functional pharyngeal valve, as evidenced by her ability to snort. Her speech is improving greatly.

Case 5.—Miss N., age 26. Complete cleft of soft and hard palates, passing through alveolar margin on the left side, and associated hare-lip. The lip had been

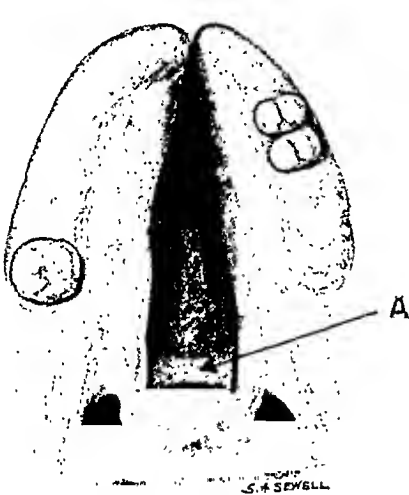


FIG. 140.—Case 5.—Before operation.
A. Ridge of Passavant.

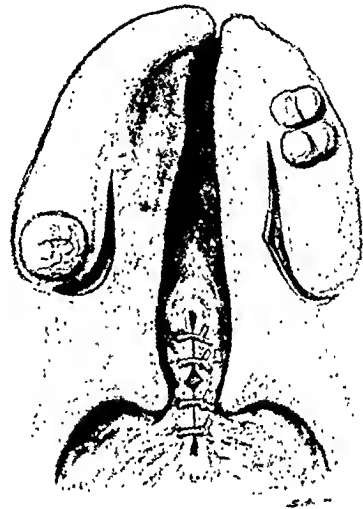


FIG. 141.—Case 5.—First stage completed. Lateral incisions made for Langenbeck-Fergusson repair.

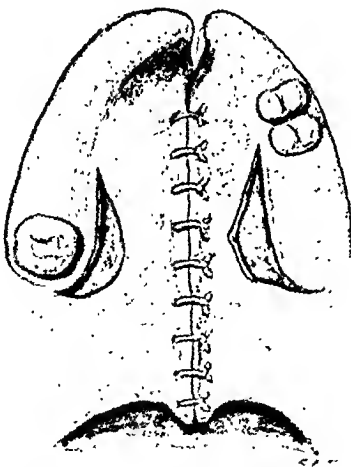


FIG. 142.—Case 5.—Completion of second stage.

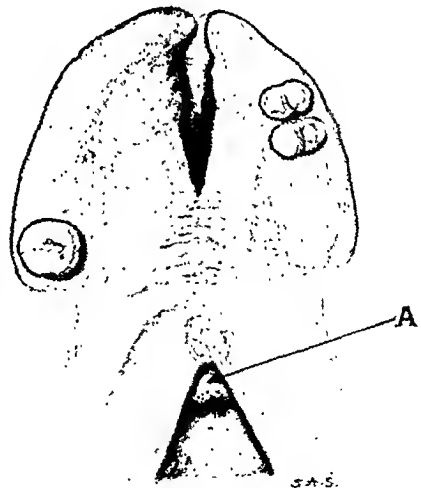


FIG. 143.—Case 5.—Seven weeks after operation.
A, Lower edge of pharyngeal ridge.

repaired in childhood. At the ages of 11 years and 12 years she underwent operations for repair of the palate. Both were complete failures, and the resultant scarring was obvious. (Evidently extensive lateral incisions had been made). Speech was very poor indeed, but was improved by wearing a plate. The levatores of the palate were poorly represented. The ridge of Passavant was very prominent during

the pronunciation of 'Ah'; its ends could be seen to pass into the soft palate. It could be seen to remain in a state of contraction throughout the whole of speech.

OPERATION (Oct. 29, 1927) (*Figs. 140, 141, 142*).—A transverse incision was made through the pharyngeal mucosa and the superior constrictor separated from the buccopharyngeal fascia. The dissection was carried as far as the salpingopharyngeal muscles. Lateral incisions, as shown in the diagram, were made in the palate to relieve tension. The palate was then repaired by the Langenbeck-Fergusson method, the two stages being done at one sitting.

Later, the anterior part of the hard palate broke down. (The patient says she remembers putting her tongue through it.) (*Fig. 143*.) The soft palate was fairly mobile, and has remained so to the present. Examination shows the lower part of the newly-formed cushion projecting below the lower margin of the soft palate.

This patient now possesses a functionally competent nasopharyngeal valve. The evidence for this lies in two directions and is quite conclusive. Firstly, she is able to snort. Secondly, fluid poured into the nose can be held at the back without any escaping into the mouth. Unless the nasopharyngeal valve were competent neither of these feats would be possible. With regard to speech, this patient has shown great improvement in the short time which has elapsed since the operation. Many of her consonants are now emitted without nasal escape. The nasopharyngeal valve being now competent, there would appear to be no reason why the remaining sounds should not become perfect by practice.

Case 6.—Elsie F., age 15. Pre-operative condition showed a very wide cleft of the soft and hard palates nearly as far as the alveolar margin. There was wide separation of the alveolar margins, and very little tissue either in the hard or soft palate. Movement of the levator muscles was very poor, and no ridge of Passavant could be seen on the pharyngeal wall. Speech was absolutely unintelligible. This case was regarded as one of the worst possible to tackle.

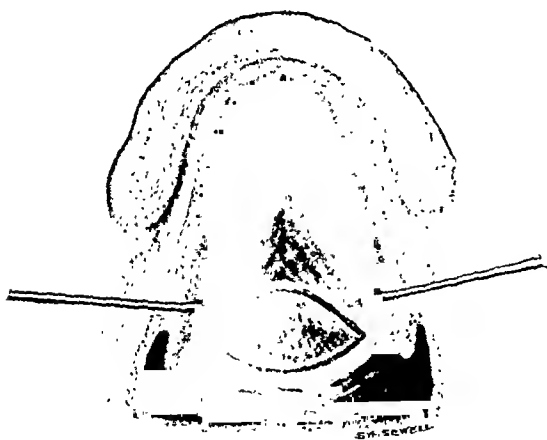


FIG. 144.—Case 6.—First stage preparatory to suture.

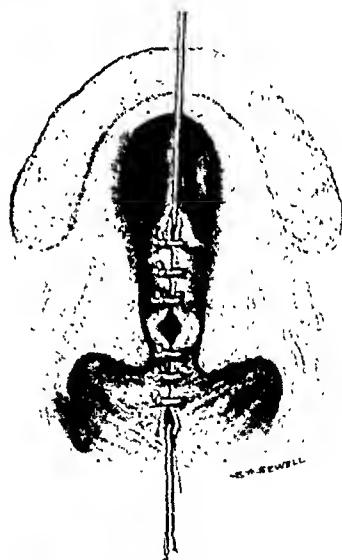


FIG. 145.—Case 6.—First stage completed. Note the approximation of the halves of the soft palate.

FIRST OPERATION (Oct. 29, 1927).—An extensive transverse incision was made in the pharynx, reaching as far as the salpingopharyngeal folds on each side. The superior constrictor was freely separated and then sutured in a vertical direction (*Figs. 144, 145*). A tension suture was passed through the faucial pillars. The two halves of the uvula almost touched at the finish.

Recovery after operation was uninterrupted and no complaint of pain was made. The wound appeared to break down about the fifth day, and it was expected that a complete failure would result. There was an intense hyperæmia of the pharyngeal

mucosa, but on this clearing up it was seen that a considerable pharyngeal cushion remained.

SECOND OPERATION (Nov. 25, 1927).—Repair by the Langenbeck-Fergusson method, care being taken to divide the hamular processes. Short lateral incisions passing around behind the teeth of both sides were made. The whole proceedings were very much embarrassed by alarming hæmorrhage, and it was at one time felt that continuation of the operation should be postponed. Fortunately it was found possible to complete the operation. It was thought that the hæmorrhage was in some way associated with the intense hyperæmia following the first stage. A silkworm suture through the tongue was necessary to prevent asphyxiation. Recovery was uninterrupted, except for the extreme discomfort associated with inability to breathe through the nose. A hole, as depicted in *Fig. 146*, was present fifteen days after the operation.

It is of interest to note that during this girl's stay in hospital after the second stage, the other patients in the ward used to complain of being kept awake by her snoring.

The soft palate in this case, as might be expected, was very rigid, but that a functional valve was formed was evidenced by her ability to snort. Speech as yet shows little improvement.

Case 7.—Norman M., age 9. Presented a cleft of soft and hard palates as far as the alveolar margin (*Fig. 147*). A cleft, passing through the margin, had closed in the process of growth. The ridge of Passavant was poorly represented and movement in the levatores of the palate bad. There was a double hare-lip. Speech was unintelligible.

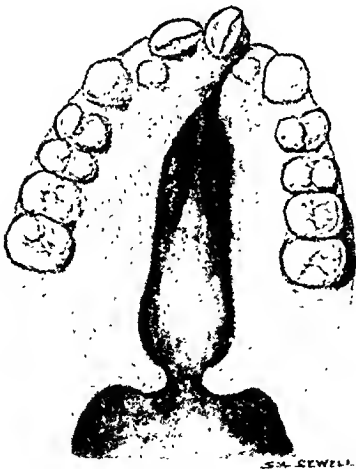


FIG. 147.—Case 7. Before operation.

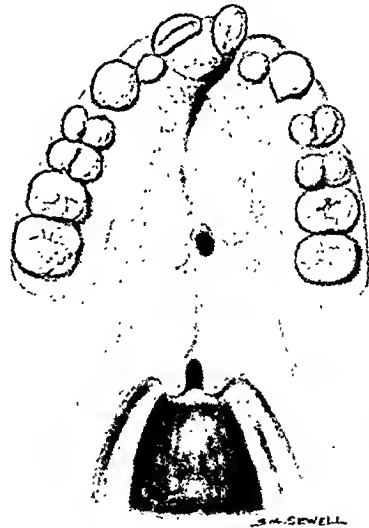


FIG. 148.—Case 7. Condition five weeks after second operation.

FIRST OPERATION (Aug. 11, 1927).—*1st Stage:* A very inadequate pharyngeal plastic operation was done. The salpingopharyngeal folds were not sutured. Very little in the way of pharyngeal cushion remained after healing of the wound. The hare-lip was also repaired.

SECOND OPERATION (Nov. 6, 1927). *1st and 2nd stages:* A long transverse incision was made in the pharynx, and the salpingopharyngeal folds were divided. The wound was sutured vertically. The operation was carried out in such a way as to obviate any possibility of error in the formation of a pharyngeal cushion. The palate was then repaired by the J. method.

This was the first case in which are must be taken to prevent asphyxiation before recovery from the anaesthetic. Recovery from the operation was uninterrupted. A small hole formed at the junction of the hard and soft palates, and one of the sutures in the uvula broke away, leaving the latter incompletely repaired (Fig. 148).

The soft palate was left relatively immobile, but the patient in spite of this was able to snort. Speech as yet shows little improvement.

Case 8.—John B., age 4. This was the case, the details of which are reported on p. 140, in which death followed thirty-six hours after performance of the first stage.

Case 9.—Wm. S., age 45. This was a complete cleft of the hard and soft palates as illustrated in the picture (Fig. 149). The cleft was not wide and the palatal arch was high. The ridge of Passavant was well developed and ran on each side into a wide lateral recess. The soft palate was mobile. Intelligence was rather below the average. Speech was in that group next to the worst, and was almost unintelligible.

FIRST OPERATION (Dec. 5, 1927).—A transverse incision was made in the superior

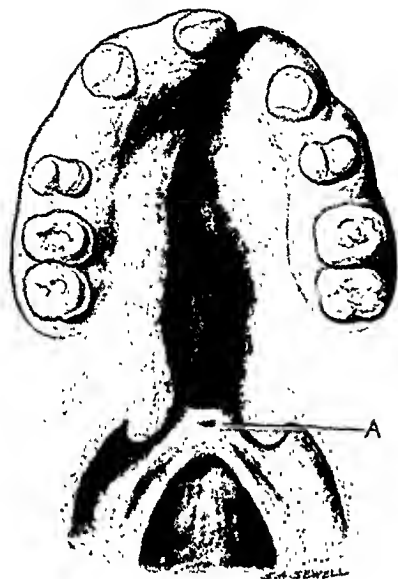


FIG. 149.—Case 9. Six days after the first stage. A is the artificial pharyngeal ridge, which has given a little in the centre. This ridge was examined six months later and no material change was noted.

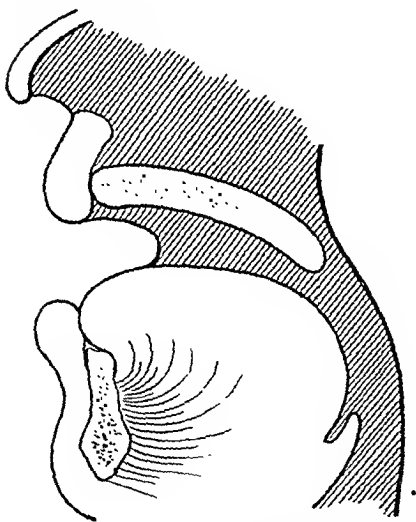


FIG. 150.—Tracing from skiagram of Case 9. Palate in resting position. After repair by combined Langenbeck-Fergusson operation and pharyngoplasty.

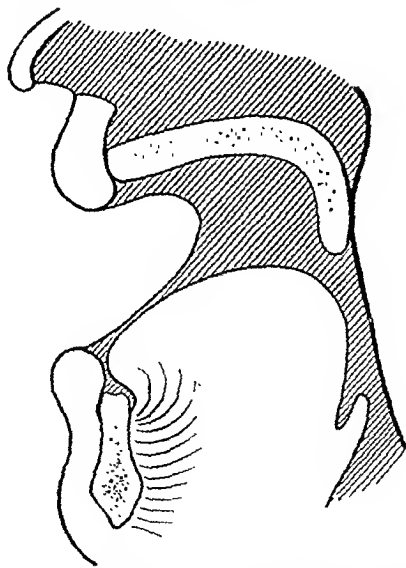


FIG. 151.—Tracing from skiagram of Case 9, after operation and during pronunciation of 'Ah.' The soft palate meets the posterior pharyngeal wall.

constrictor and the wound was closed vertically in the usual manner. Care was taken to suture the salpingopharyngeal folds, and one tension suture was fixed in the faucial pillars. At the end of six days the wound was found to have given a little in the centre; thereafter it remained stationary (*Fig. 149*).

SECOND OPERATION (Jan. 9, 1928).—The hard and soft palates were repaired by the Langenbeck-Fergusson method, the hamular processes being divided. Once again a suture through the tongue was necessary during recovery from the anæsthetic. A hole appeared at the junction of the hard and soft palates, and is now rapidly closing. A competent valve was formed, as proved by the ability to snort. Further evidence in this case lies in the fact that the pharyngeal cushion was made lower down than is the usual practice, and the lower edge of the soft palate can be seen to come in contact with this when he says 'Ah'. Skiagrams taken in the resting position of the soft palate and during the pronunciation of 'Ah' show convincing evidence of the competence of the valve (*Figs. 150, 151*). Speech at first was worse after the operation; but, even in the short time which has elapsed, improvement is beginning.*

In six of the cases mentioned above the ability of the patient to snort has been used as a proof of the fact that the soft palate comes in contact with the pharyngeal wall. To obviate any possibility of this being mistaken for stertor, the patient is always instructed to protrude the tongue and then grip it between the lips and teeth before applying the test. Many cases of cleft palate after the Langenbeck-Fergusson operation have been examined. Only one was able to snort: she was one of the rare cases of perfect speech result. The cleft was of the soft palate only.

CONCLUSIONS.

An attempt has been made to stress the importance of the formation of a pharyngeal valve in the sufferer from cleft palate, and it is believed to be impossible to restore normal speech unless this is first done. By the method described and applied in eight cases (excluding one death) this has been accomplished in six. In the first two cases the attempt failed for the good reason that the technique of the procedure was not fully understood and the anatomical points of importance could not be anticipated. Practice of the method has assured increased speed and certainty in the attainment of the result, and it is felt that future cases will be approached with a confidence that, at any rate in a large percentage, a functional valve will be formed. That this result cannot fail to reflect itself in improved speech must be obvious to all who have made a study of the subject.

In the discussion of the results of the procedure recommended, little mention has been made of the effects upon speech because of the short period of time which has elapsed since the earliest of the cases was submitted to operation. But there is abundant evidence, even at this early date, to make one sanguine of the issue. No claim is made to have found the solution of the problem in cleft palate, because all the surgeon can do is to provide a

* As mentioned above, this patient was able to snort after repair of the palate. He maintained this faculty for some weeks, when it gradually disappeared. Its loss was due entirely to contraction of scar tissue around the hole at the junction of the hard and soft palates. This gradually pulled the palate away from the posterior pharyngeal wall, leaving the artificial ridge completely exposed. There would appear to be no doubt as to the permanence of the ridge, which has not changed since the condition depicted in *Fig. 149*.

physiological palatal mechanism as a basis for the subsequent acquisition of speech. Perseverance, ambition, and shame are not yet included in the list of conditions amenable to plastic surgery, and so much depends on these abstract qualities, together with the intelligent guidance of a good teacher, that any claim to infallibility would be a claim to the impossible. Dr. Victor Veau² rightly asks, "Shall we ever know how to operate upon a cleft palate with as much security as upon inguinal hernia?"

The number of cases recorded is small, and all are of recent date. None of them was specially chosen for the purpose of swelling statistical columns; in fact, as a collection of cases upon which to attempt a new operation, they are as bad a lot as could be fairly expected. Two of them had had operations in earlier life (*Cases 1 and 5*), with consequent scarring. One had a wide cleft which might well have been adjudged inoperable (*Case 6*). In two (*Cases 6 and 7*) the muscle of the palate and pharynx was poorly represented and almost immobile even before operation, a fact which reflected itself in a relatively immobile palate afterwards; yet both of these cases were able to snort.

In conclusion a few questions arise in connection with the treatment suggested.

1. Will the cushion formed on the pharyngeal wall persist? Time and experience alone will provide the answer to this. In the meantime it may be stated that, so far as human observation can determine, there is no change in this respect over a period of months.

2. Is the method applicable to infants? The only child on whom the operation was essayed died. There appears to be no reason why, with the experience gained, the extension of the method to infants should not be attempted.

3. To what other uses may it be applied? There are large numbers of cases who have submitted to the Langenbeck-Fergusson procedure with disappointing results. The difficulties in the way of performing pharyngoplasty after a Langenbeck-Fergusson operation are not insuperable, and it is intended to try the method on the first opportunity.

4. With regard to adenoids. In none of the above cases was it necessary to consider this question, but it is obviously impossible to perform a reasonably neat and deliberate operation unless adenoids are first removed.

It gives me great pleasure to acknowledge the assistance I have received from many sources. Sir Arthur Keith and Dr. Victor Veau have kindly placed specimens at my disposal. Mr. James Whillis, Demonstrator of Anatomy in the Durham University College of Medicine, has assisted me to elucidate points in the superior constrictor of the pharynx. Professor R. B. Green has been generous in his supply of material and advice. To Professor G. Grey Turner I tender my grateful thanks for advice and criticism, and for allowing me to examine his cases. I am deeply indebted to Mr. T. W. Moles, M.A., for the time and patience he has spent in the speech-training of my cases.

REFERENCES.

¹ PASSAVANT, GUSTAV, *Virchow's Arch.*, 1869, xlvii.

² Discussion on Cleft Palate, Royal Society of Medicine, 1927, June.

SPECIAL ARTICLES
ON SURGICAL TECHNIQUE.

**DIRECT SKELETAL TRACTION IN THE TREATMENT
OF FRACTURES.**

BY E. W. HEY GROVES,

PROFESSOR OF SURGERY, UNIVERSITY OF BRISTOL.

IT is now a generation ago since Codivilla first suggested the application of extending force to the fractured limb by means of a nail which was driven into the distal fragment of bone. This method, having been elaborated by Steinmann, was quickly adopted in Germany, Switzerland, and Holland, and more slowly in other countries. It has never been of general application in this country, although at the close of the War period, and especially under the influence of Pearson, it was used more frequently than any other method in the treatment of open fractures of the femur.

GENERAL PRINCIPLES.

There are two main advantages of skeletal traction in the treatment of fractures. First, it is by far the most efficient and reliable method for reducing shortening, or overlapping, especially if this has been of some duration. I would go so far as to say that, provided bony union of a fracture has not taken place, there is no malposition of a broken long bone which cannot be fully corrected or over-corrected by adequate skeletal traction, in regard to the full length and proper axis of the limb. Fractured femurs with three to five inches of shortening can be restored to full length, usually within a few days. And further, bad cases of mal-union can be similarly corrected if a preliminary osteotomy be done. In the second place, skeletal traction leaves the whole of the limb and the joints free for dressing of wounds, massage, and joint movements. This is of very great value in septic or open fractures, and in all cases when union is slow and the treatment necessarily prolonged.

Appliances.—Before describing the technique which I have found to be the most satisfactory, it may be worth while to discuss very briefly the different appliances which have been used by various workers for skeletal traction. These are represented by the following list: Transfixion pin; nails driven into the sides of the bone; ice-tongs calipers; horseshoe clamp with screws; os-calais stirrup; silk or wire loops.

The *transfixion pin*, which goes right through the bone. I have always regarded as the best of all these devices, and after trying all the others I have come back to this, as the one which by reason of its reliability and simplicity

is the most satisfactory. The objection which has been urged against it is the liability to produce septic sinuses leading down to the bone. If due precautions are used, this drawback is rarely encountered, and an occasional sinus can easily be cured by simple curetting.

Nails driven into the side of the bone have nothing to recommend them. They are merely an improvised method used when the proper transfixion pin or tongs are not at hand. It is clear that two nails driven into the femoral condyles, for instance, cannot support a big traction force and will soon be dragged out of line by leverage.

The *ice-tongs caliper* is perhaps the method most widely used by those who employ skeletal traction, and it has been designed in a number of different shapes in order to ensure or to prevent the penetration of the outer shell of the bone by the points of the caliper. It is claimed for the caliper that it is superior to the transfixion pin, because it only holds the outer shell of bone and therefore does not cause a chronic sinus after its removal. But it is certainly more difficult to use for bad cases where a big traction force is necessary. Its correct adjustment in order to get the right degree of bone penetration is a difficulty, and this adjustment is liable to become disturbed during prolonged treatment. But this is perhaps a matter of personal predilection rather than one which involves any vital principle. Certainly the caliper requires to be supervised by nurses who are accustomed to its use, whereas the transfixion pin is practically fool-proof.

The various types of *horseshoe clamps* are open to exactly the same criticism as the calipers, and therefore no more need be said about them.

The *stirrup*, invented by Finocchietto, which passes over the upper surface of the calcaneum between the tendo Achillis and the ankle, has the advantage of not requiring any bone penetration. But for this same reason, as it only goes through the soft parts, it is liable to slight movements, and this causes it to be much more painful than the rigidly placed transfixion pin.

Silk or wire suture loops have been described and used, chiefly for traction of the olecranon and the calcaneum. They are both neat and effective, but they do not present any real advantage over the transfixion pin, and if used for the heel bone they have the drawback of causing a wound on the plantar surface of the heel which may be troublesome.

PIN TRACTION APPLIED TO THE FEMUR.

It is not necessary to discuss the indications for skeletal traction of the fractured femur. The majority of surgeons will agree that it is indicated for all those cases neglected or insufficiently treated at the time of injury in which gross displacement has occurred with much overlapping and shortening. Also for cases of a later date where actual mal-union in faulty position has taken place. There is no general agreement, however, as to the use of this method in cases of recent fracture.

The apparatus—pin, handle, and traction stirrup—are made in different forms, but these varieties are of no essential importance. For the femur I use a pin 6 in. long and $\frac{3}{16}$ in. thick, with a bradawl point at one end and a flat bearing surface at the other which fits into a handle (*Fig. 152*). The patient

is always given a general anæsthetic, not only for the transfixion, but because the most important act of traction is the pull made upon the limb immediately afterwards, and for this full muscle relaxation is necessary. No doubt spinal anæsthesia would achieve the same object, but I have not employed this method myself.

The patient being anæsthetized and the skin prepared, the pin held in the handle is thrust into the soft parts without making a special incision for it, the integument being strained upwards towards the fracture whilst the transfixion takes place. The object of the latter detail is to lessen the tension on the skin when traction is applied; in the same way the pin, when it has gone through the bone, is made to emerge through the skin on the other side of the limb, the skin being pulled upwards. The site of transfixion should not be through the condyles, but well above them, through the lower part of the femoral shaft (*Fig. 153, B*). This is for two reasons: the cancellous tissue of the condyles is soft and gives way before the pressure

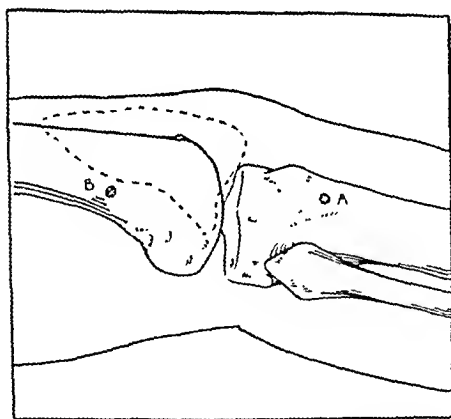


FIG. 153.—B and A. Points of transfixion of femur and tibia. Dotted lines show the limit of synovial membrane. (From "*Modern Methods of Treating Fractures*".)

bandage. The metal tractor has two small pulley wheels on its transverse bar, which now lies exposed below the knee-joint (*Figs. 154, 155*).

The transfixion having been accomplished and the tractor put in position, full manual traction is applied to the limb, and if any faulty union has already taken place this is broken down. The patient is put in a bed provided with a metal overhead bar to which the limb is slung, the knee being flexed to about 120°. The upright member of the overhead beam is provided with a

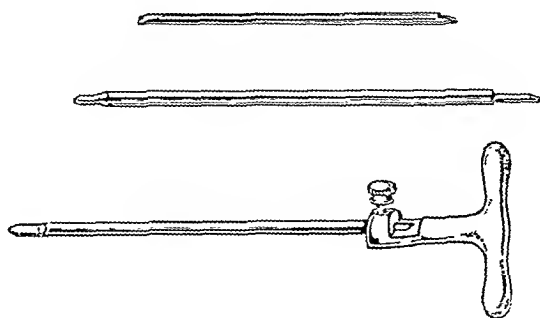


FIG. 152.—Transfixion pins and handle ($\frac{1}{4}$ actual size). (From the author's "*Modern Methods of Treating Fractures*," John Wright & Sons Ltd., Bristol.)

of the pin, whereas the dense shaft will allow a big traction force without the pin working loose; and further, the high transfixion site avoids all danger of wounding the synovial sac of the knee-joint.

The limb having been transfixed, there is about 1 or 1½ in. of bare pin projecting on each side. This is covered with rubber tubing, which gives a non-slipping grip to the metal tractor, and also serves to protect the sharp ends of the pin, preventing them from scratching the patient or the nurse. Some layers of dressing are applied, with a bandage, and then the metal tractor shaped like a U is put on and kept in place by further dressing and

double pulley which can be adjusted at any height. A cord is tied to the fixed upright and passed alternately round each of the pulleys, two at the bed-foot and two at the knee, in the way shown in *Fig. 155*; finally, as it drops from the second pulley at the fixed upright it has the weight attached to it. This weight will vary from 5 to 15 lb. according to the type of patient and of fracture, giving with the fourfold pulley multiplication a traction force

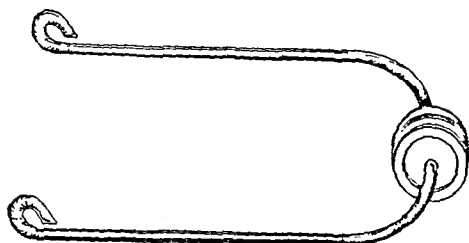


FIG. 154.—U metal tractor with pulley wheels.

of 20 to 60 lb. The smaller weights are sufficient for fractures of recent origin or for small thin patients, whilst the larger ones are needed for old fractures in muscular subjects. In any case it is much better to begin with too big than too small a weight, because then the reposition of the overlapping fragments is more quickly accomplished, and directly this is done the weight can be diminished. The reduction of

the weight is most conveniently done by altering the attachment of the cord, so as to use only 3 or 2 or 1 pulley. Generally the weight is thus reduced by one quarter at the end of one week, and by one half at the end of three weeks. If enough weight is used, then full reduction, and over-reduction, takes place; the fractured ends become disengaged from each other and

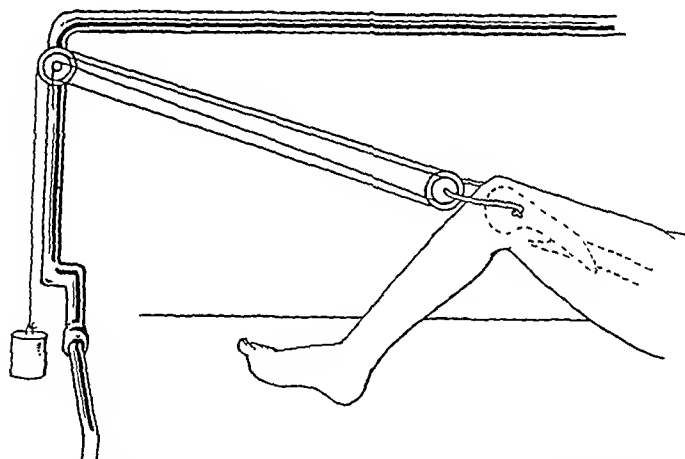


FIG. 155.—Multiplying pulleys. The pulleys are here drawn different sizes in order to show all four cords. Actually they are both the same size. (From the author's article on 'Fractures of the Long Bones', in the "Index of Treatment", John Wright & Sons Ltd., Bristol.)

from soft tissues, and can then be slowly allowed to assume the correct apposition.

In using these big weights efficient counter-extension is very important. The foot of the bed should be raised on 12-in. blocks and a padded perineal band passed round each thigh and tied to the corresponding upper post of the bed.

Usually the pin traction can be discontinued at the end of four to six weeks, and then its place may be taken by adhesive plaster traction or the use of a walking caliper. In the above description I have only spoken of the limb as being slung in a semi-flexed position of the hip and knee to an overhead beam. This is undoubtedly the technique which gives the traction force the fullest efficiency. But after a few days, before the end of the first week, the leg will have come to be of full length, and it is convenient then to apply a Thomas splint with a hinged leg-piece and foot-piece as a rest for the whole limb, without interfering with the traction.

Quite apart from special complications, pin traction of the fractured femur requires to be modified for two special positions of the fracture. In those high up, through the trochanters, it is essential to secure full abduction, and this can be done most conveniently by applying the perineal counter-extension to the sound side only, thus drawing up that side of the pelvis, whilst the broken limb is swung outwards to the side of the bed. In fractures of the lower third, or those involving the knee-joint, the transfixion must be made through the tibia and not the femur. A short pin, 4 in. long and $\frac{1}{8}$ in. thick, is driven through the crest of the tibia just below the tubercle (see Fig. 153, A). Through such a pin very efficient traction can be made, as it pulls upon the actual bony insertion of the extensor and flexor muscle groups of the knee. Another point in favour of the tibial transfixion site is that the pin can be left in place for a long time—six or eight weeks—and for this reason it is well adapted for the treatment of septic open fractures.

FRACTURES OF THE TIBIA AND FIBULA.

Skeletal traction is not required nearly so often in fractures of the leg as in those of the thigh; but nevertheless it is of the utmost value in those cases where it is indicated. Fractures of the tibia and fibula never show the same tendency to great overlapping as do those of the femur. Often the fracture in the early days has only about $\frac{3}{4}$ in. overlap and looks as if simple manipulation would serve to effect reposition; but even under an anæsthetic it often proves to be so difficult that this remains now as the common type of fracture to be subjected to an open plating operation. But it is just in such an obstinate fracture that skeletal traction will prove to be quite a simple remedy.

The transfixion is by the small short pin, which is passed through the posterior process of the calcaneum as far back as possible so as to afford a counter-traction to the pull of the tendo Achillis (Fig. 156). The leg must be put up in some form of sling, and I find the short cradle splint (Fig. 157) most convenient for this purpose. The weights used are about half those mentioned in the case of the femur—i.e., 10 to 30 lb.—and usually two pulleys are sufficient.

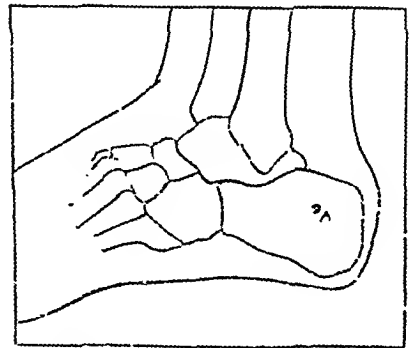


FIG. 156.—Point of transfixion for os calcis. (From "*Modern Methods of Treating Fractures*".)

Pin traction through the calcaneum has two special advantages in addition to those of efficiency: the foot is kept in dorsiflexion, because all the pull is made on the heel; and as the traction is made through the ligaments of the ankle-joint, the latter is kept open and supple.

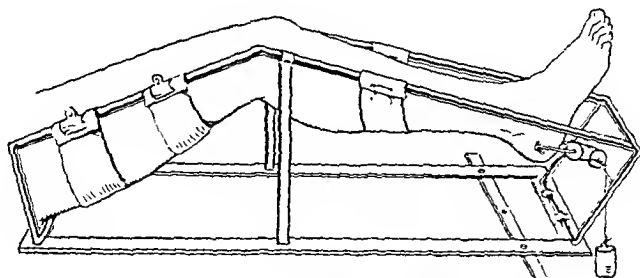


FIG. 157.—Author's short cradle splint.



FIGS. 158 and 159.—Skiagraphs of fracture-dislocation of tibia and fibula, before and after treatment.

In two types of case it is better to use a big pin and to transfix the malleoli rather than the heel bone. These are fractures of the upper third of the leg bones, and those fracture-dislocations in which the inferior tibio-fibular joint has been damaged, with marked separation of the two bones (*Figs. 158, 159*).

FRACTURES OF THE LOWER END OF THE HUMERUS.

In the upper limb, skeletal traction is very seldom required, but there is one application which may be of great value. This is for the various types of fracture of the humerus just above or into the elbow-joint, especially when there is great swelling of the elbow which contra-indicates any forced flexion. In such a case the arm is slung to an overhead support with the

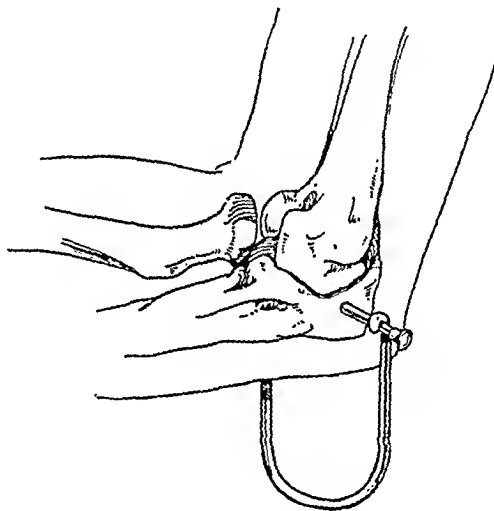


FIG. 160.—Extension of humerus by transfixion of olecranon. (*From "Modern Methods of Treating Fractures".*)

elbow at a right angle, the olecranon is transfixed with a slender short pin (*Fig. 160*), and a weight of about 10 lb. is attached. In this way the displacement of the fracture can be reduced fully, whilst the limb is kept in the best possible position for the restoration of the circulation and the preservation of the elbow movements.

DIRECT SKELETAL ADJUSTMENT OTHER THAN TRACTION.

It not infrequently happens that after skeletal traction has fully reduced or over-reduced overlapping, there still remains some lateral displacement which it is desirable to correct. In the forearm bones lateral displacement is usually the most important element in the deformity, and in the leg bones the displacement of a spiral fracture is notoriously difficult to adjust. This type of lateral displacement can usually be brought into perfect position by means of a simple open exposure, when the bones can be made to interlock. If they will not remain in close contact, then by means of a metal bone bradawl each fragment is partly transfixed and held in position whilst the

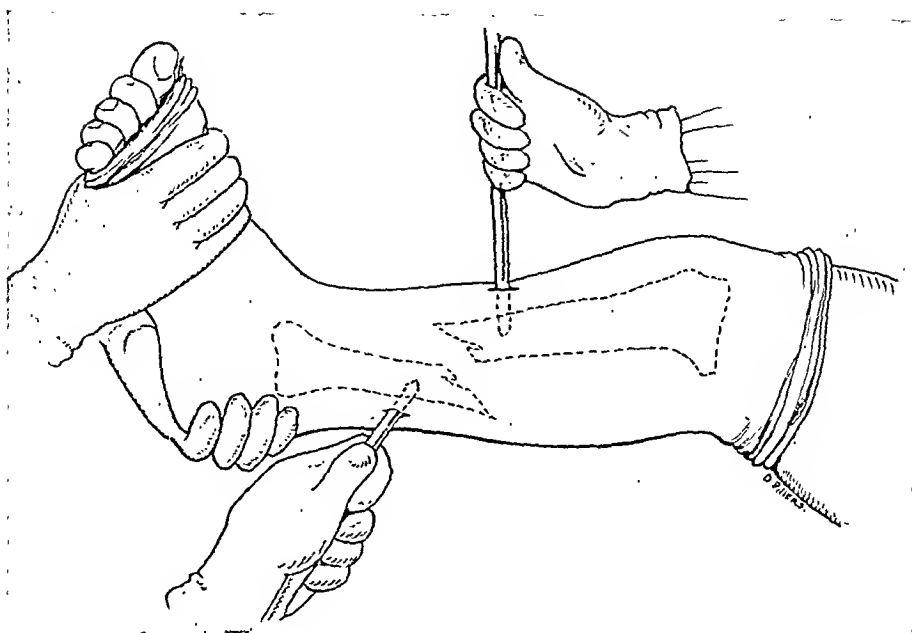


FIG. 161.—Reduction of lateral displacement by the use of bradawls: before reduction.

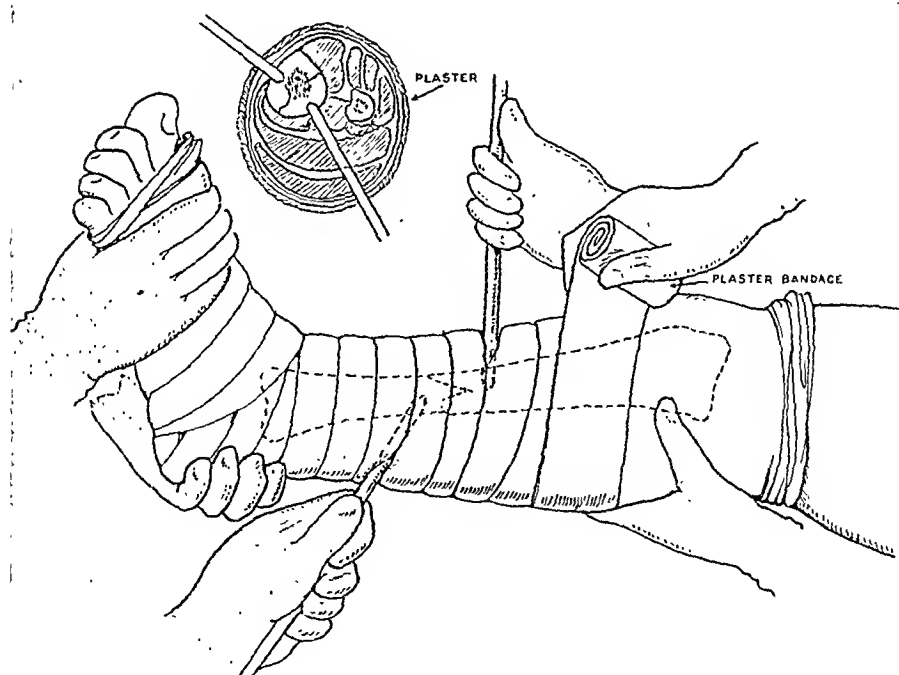


FIG. 162.—The bradawl operation: after reduction and fixation in plaster.

wound is closed and the limb is put in plaster (*Figs. 161, 162*). The plaster surrounds the projecting shafts of the bradawls, which are left in place for



FIG. 163.—Skiagram of fractured leg bones before bradawl operation.



FIG. 164.—The same case four weeks after operation.

twenty-four hours. If this method is used, it is noteworthy how rapid is the natural healing by callus as compared with the delayed union which generally follows the application of a plate and screws (*Figs. 163, 164*).

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF LARGE SINGLE (NON-PARASITIC) CYST OF THE SPLEEN.

By A. W. SANDERS,

SURGEON TO THE PRETORIA HOSPITAL.

IN a paper published in *Surgery, Gynecology and Obstetrics* in November, 1927, by Emil Novak, of Baltimore, it is stated that only seventy-four cases of non-parasitic cyst of the spleen have been recorded. It would therefore seem worth while to place on record another. This case would appear to come under the heading of 'traumatic cysts', judging by its appearance and the pathological report, although there was no history of any injury.

HISTORY.—The patient, a girl of 15, who had been perfectly well recently, and who had never had any illness of note, was seized one morning with acute abdominal pain not accurately localized but referred to the lower abdomen. Her temperature rose to 102°, and she looked at first pale and pinched but later on was flushed. The pain was of brief duration and had quite passed off in an hour, the temperature having by that time fallen to normal. No tenderness or rigidity was to be made out, but one found what appeared to be a definite enlargement of the spleen. This organ could be felt extending about an inch and a half below the costal margin, occasioning a slight fullness and prominence in the left hypochondriac region. The swelling was not tender, and, indeed, there was nothing to indicate that it was in any way associated with the severe pain and rise of temperature. There was no history of any injury recent or remote. A cytological examination of the blood showed nothing abnormal.

The patient was kept under observation and examined from time to time, and it was found that the tumour was enlarging slowly, but no symptoms occurred to give rise to any anxiety. An X-ray examination gave no information as to the nature of the tumour.

Some weeks after the discovery of the tumour the patient was examined by Professor Ritchie Thompson, of Johannesburg, who found that it could be displaced forwards and to the right as far as the mid-line. It was then felt to be rounded in shape. No distinct notch could be made out, but it was evidently attached to the splenic region by a definite pedicle. The tumour did not occasion the least pain or discomfort, and the girl looked and felt

perfectly well. Beyond limiting her freedom as to active exercise, particularly gymnastic, no precautions were adopted. The possibility of the tumour being of renal origin was considered. There was an entire absence of renal symptoms, and the mobility and the situation of the tumour immediately below the costal margin and beneath the abdominal wall made its splenic origin seem much more probable. The steadily increasing size of the tumour impelled a decision in favour of operation. Professor Ritchie Thompson kindly assisted.

OPERATION.—The abdomen having been opened by a vertical incision about 6 in. long, 1 in. to the left of the mid-line, the tumour was readily exposed. The upper part of the spleen appeared normal in shape and consistency, but the lower half was occupied by a cyst about 4 inches in diameter. The cyst was so thin in places that the dark fluid contents showed clearly through it; in other places it was thick, white, and opaque, as though the peritoneal covering had been the seat of an inflammatory change due to friction against the abdominal wall. The cyst extended deeply into the substance of the spleen—in fact well down to the hilum. The fluid contents were evacuated and found to be dark brown in colour, with glistening particles, possibly fat globules, or, as it was thought, cholesterin crystals; but unfortunately no proper examination of the fluid was made. The cyst was for the most part about $\frac{1}{16}$ in. thick, tough and fibrous in character, and of a reticulated appearance in the deeper or imbedded portion. An attempt being made to shell out the more superficial portion showed that the wall was firmly attached to the splenic tissue, and that to remove it in this manner would cause much hæmorrhage and leave a very ragged damaged surface. Being loth to remove the entire spleen if it could be helped, it was decided to attempt the resection of the lower portion containing the cyst. The hilum in the neighbourhood of the cyst was clamped with two artery forceps, and the portion above grasped in the assistant's fingers. The cyst with a wedge of splenic tissue was then excised. The vessels at the hilum that had been clamped were ligatured. The raw surfaces of the spleen were now brought firmly face to face with mattress sutures of coarse catgut, a few strands of the same sized gut being caught under the loops of the stitch so as to serve as a button and prevent the suture cutting through. The approximated edges were then oversewn with a continuous catgut stitch, and the wound was entirely covered with a flap of the great omentum turned up from below and firmly sewn down to the spleen stump.

The amount of blood lost was but slight; the patient stood the operation well, made an uninterrupted recovery, and has had no symptoms since.

Dr. Pijper, who kindly examined the cyst wall, reported that it was composed of fibrous tissue, and had no epithelial or endothelial lining.

It may be questioned whether it was wise to attempt the saving of a portion of the spleen, and it may be argued that splenectomy would have been a safer procedure; but possibly the last word on the functions of this organ has not yet been said, and to retain half a healthy spleen may be an advantage to the patient.

A CASE OF MULTIPLE CHONDROMATA.

By J. W. THOMSON,

HONORARY SURGEON TO THE CLAYTON HOSPITAL, WAKEFIELD.

THE patient, I. K., was a man of 23 years. At the age of 2 it was noticed that there were swellings on the fingers of the left hand and wrist. These swellings increased in size and became painful. When he was 13 years old the left index finger was amputated, and a month later, because of pain and deformity, the left arm was removed above the elbow.

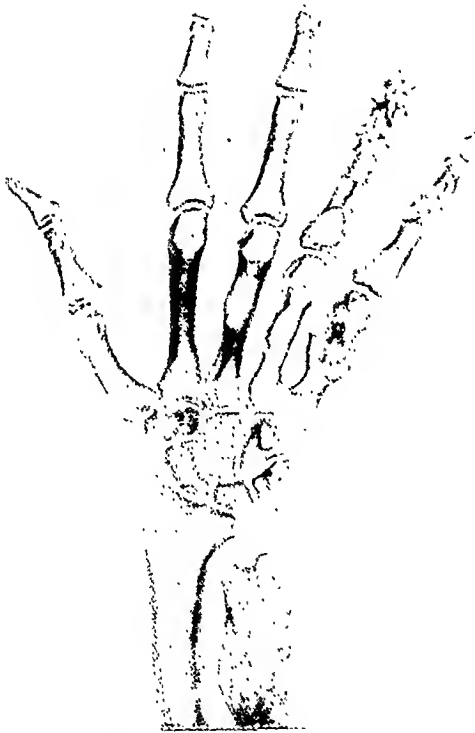


FIG. 166.—Right hand, from behind, at the age of 23.

Recently a complete radiological examination was made, and disclosed multiple tumours in the right hand, chiefly in the ring and little fingers and in the lower end of the ulna; also in the little toe of the right foot. None of these growths cause any pain or inconvenience. Section of one tumour from the finger shows it to be a pure chondroma.



FIG. 165.—Left hand, viewed from in front, at the age of 13. Almost all the phalanges, the metacarpals, and the lower ends of the radius and ulna are the seat of tumour formation.



FIG. 167.—Right foot, at the age of 23.

A CASE OF SARCOMA OF THE ANUS.

By R. J. WILLAN, M.V.O.

HONORARY SURGEON AND LECTURER IN CLINICAL SURGERY TO THE ROYAL VICTORIA INFIRMARY,
NEWCASTLE-UPON-TYNE.

WILLIAM R., age 43, a waggon-man, was admitted on Dec. 8, 1926, to my wards at the Royal Victoria Infirmary, Newcastle-on-Tyne, with a diagnosis of double irreducible femoral herniæ. He complained of pain in the left groin which he had had for the previous three days. He had noticed a right groin swelling fourteen days, and a left groin swelling three days, before admission. There had been no vomiting or nausea; his bowels were perfectly regular. For years he had had eczema of the right leg. For the previous three years he had suffered from 'piles', which had recently become much worse; there had never been any bleeding. He had lost flesh.

On admission he looked anæmic. In each groin there was a rounded swelling the size of a hen's egg; each swelling was hard, smooth-surfaced,



FIG. 168.—Primary sarcomatous hemorrhoid laid open, with anal skin (A) adjacent.



FIG. 169.—Secondary sarcomatous lymphatic gland of groin laid open.

had a well-defined edge, and was firmly adherent both to the overlying skin and the deeper structures. Neither swelling had any impulse on coughing. He had thrombosed external hæmorrhoids. One of the posterior hæmorrhoids was hard; it was ulcerated on its inner surface, there being growth in excess of destruction in the ulcer. He had dermatitis of the right leg. Nothing abnormal could be found in his abdomen or chest.

During a short period while he was kept under observation both the anal ulceration and the groin swellings visible increased in size. I therefore excised the ulcerating hæmorrhoid and also the huge right groin lymphatic gland (*Figs. 168, 169*). His wound healed, and he left hospital a fortnight later.

Sections were sent to Professor Stuart MacDonald, who reported the tumour to be a small-spindle-celled sarcoma (*Figs. 170, 171*).

AFTER-PROGRESS.—His doctor, Dr. Aicken, of Ferryhill, informs me that "the patient, wasted to a skeleton, died from general dissemination of growth

ten weeks after he left hospital. All the time he suffered considerable pain, for which morphia had little or no effect. He also suffered from retention of urine”.



FIG. 170.—Low power view of anal growth. ($\times 90$.)

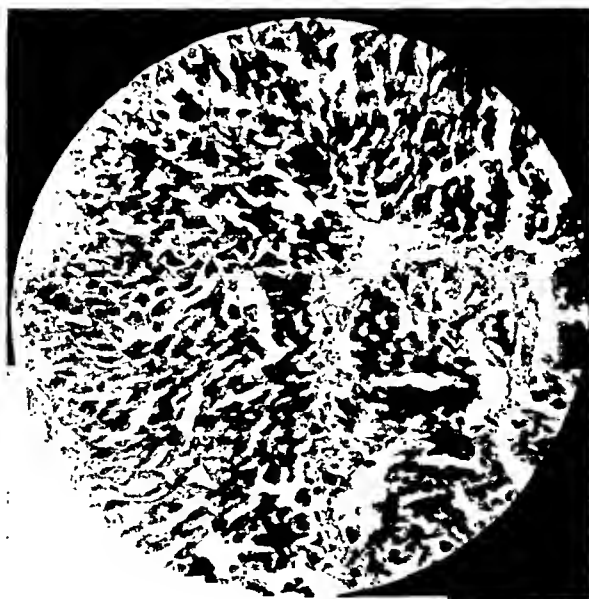


FIG. 171.—Higher power view of anal growth. ($\times 280$.)

I am indebted to Professor Stuart MacDonald for the pathological report and for the microphotographs.

A TUMOUR OF THE CAROTID BODY.

By FRANK HARVEY, LONDON.

TUMOURS of the carotid body are rare. Clinically, they are recognized as swellings in the neck about the level of the thyroid cartilage, lying either under the sternomastoid muscle or just internal to its anterior border. The swelling is generally regarded as a tuberculous gland, and accurate diagnosis is seldom made; it lies between tuberculous gland, pharyngeal diverticulum, aneurysm of the carotids, adenoma of the thyroid gland, and a branchial cyst. Shipley and Lynn¹ collected and tabulated the reported cases in 1916; and Cahill and Taylor² reported a case in 1917. Since then various cases have been put on record, the latest being that of J. E. H. Roberts, reported by M. G. Fitzgerald.³

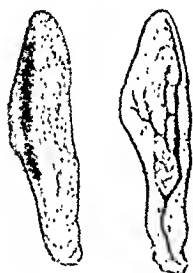


FIG. 172.—Vertical section of collapsed cyst, showing smooth outer wall and irregular inner surface. (Natural size.)

The following case is that of a lady, age 32 years, whom I saw in consultation with Dr. C. Vere Nicoll, of Frensham, Surrey, on Jan. 16, 1927.

HISTORY.—For about a year the patient had noticed a small swelling on the right side of the neck. During the last few weeks and prior to my seeing her the swelling had increased rather rapidly in size, and pain had been experienced, referred to the right ear, shoulder, and clavicular regions. The patient stated that she had never had a severe illness, and had always enjoyed good health.

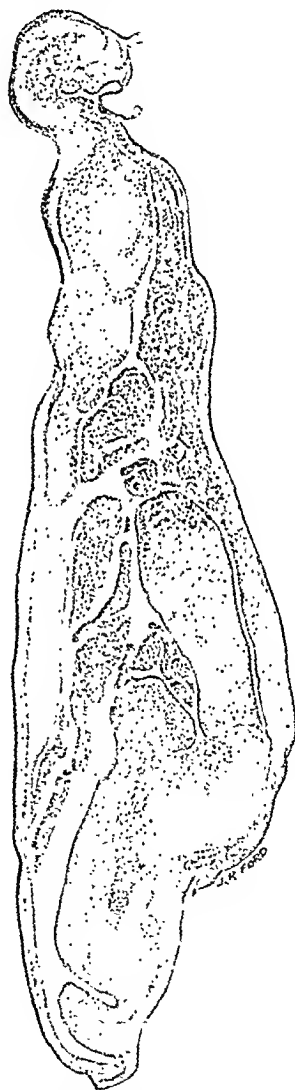


FIG. 173.—Section showing patches of pigment on the inner cyst wall. ($\times 5$.)

ON EXAMINATION.—The patient was a healthy-looking woman. Under the middle of the right sternomastoid muscle could be felt a smooth, tense, non-pulsatile swelling, about the size of a large grape. Mobility was limited. There was no enlargement of the thyroid gland, and the swelling was entirely separate from it. Also there was no movement or increase in size of the swelling when the patient swallowed. No enlarged glands could be palpated on either side of the neck. Auscultation over the swelling was negative.

OPERATION (Jan. 20, 1927).—A transverse incision was made in the natural crease of the neck at the level of the middle of the anterior border



FIG. 174.—Microscopic section showing numerous cells containing blood pigment. ($\times 65$)

of the right sternomastoid muscle, which was defined and retracted outwards, exposing the tumour. The tumour was dark purple in colour, and was projecting forwards and outwards.

The internal jugular vein was displaced outwards by the swelling. By blunt dissection the tumour was freed from the surrounding structures and was found to arise from the bifurcation of the right common carotid artery. Several moderate-sized vessels from both the external and internal carotid arteries entered the tumour. These were ligatured and the tumour was

removed. The wound was closed without drainage. The patient made an uneventful recovery, and has not since complained of any further discomfort in the neck.

PATHOLOGICAL REPORT.—The specimen (*Fig. 172*) was examined by Dr. A. Osborne, who reports as follows:—

“The cyst is about an inch in diameter. The cavity of the cyst is irregular in shape and the walls are pigmented (*Fig. 173*). Histologically,



FIG. 175.—Microscopic section showing distortion of the polygonal cells, together with fibrosis. ($\times 65$.)

the features are those of a remote hæmorrhage in the carotid body (*Fig. 174*). There is much fibrosis and distortion of the polygonal cells of the carotid gland (*Fig. 175*), and in the fibrous stroma are numerous cells containing blood pigment. The tissue is innocent.”

REFERENCES.

- ¹ SHIPLEY and LYNN, *Jour. Amer. Med. Assoc.*, 1916, lxvi, 1602.
- ² CAHILL and TAYLOR, *Ibid.*, 1917, lxviii, 1898.
- ³ FITZGERALD, *St. Bart's Hosp. Jour.*, 1927, xxxiv, 62.

THE TREATMENT OF RAYNAUD'S DISEASE BY PERI-ARTERIAL SYMPATHECTOMY.

By HAMILTON BAILEY,

SURGEON TO THE DUDLEY ROAD HOSPITAL, BIRMINGHAM.

For peri-arterial sympathectomy to be efficient from a vascular standpoint, the artery upon which it is performed must be capable of active contraction and dilatation. If the vessel is rigid and calcified, it is unreasonable to expect that the operation will prove of much therapeutic value.

Even in advanced Raynaud's disease the arteries, though distinctly smaller than usual, remain soft and pliable. Here, then, is a condition in which sympathectomy should prove highly effective. A study of two cases has convinced me of the value of peri-arterial sympathectomy in this disease.

Case 1.—An able-bodied seaman, age 44, was invalided from the Royal Navy in April, 1927, with Raynaud's disease. The manifestations were typical in all respects; spasms were occurring frequently, and whilst under observation one could see his tremulous hands go blue; at the same time the fingers down to the interphalangeal joints assumed an ashen hue. The symptoms were largely bilateral, but he complained specially of his right hand, and in September, 1927, the finger-tips of this hand, particularly of the second and third digits, became black, and the skin in this region was obviously commencing to undergo dry gangrene.

On Sept. 15, 1927, peri-arterial sympathectomy was performed on the right brachial artery. The adventitia of the artery was removed for fully three and a quarter inches. At the time of the operation it was noted that the artery was distinctly smaller than normal. There was immediate relief of the symptoms on this side. When seen in November, 1927, the right hand had a normal appearance. The dead, black skin had separated, and a new nail was commencing to grow on the third finger. The patient now complained bitterly of his left hand. On further observation it was interesting to see that the typical spasms were now confined to the unoperated side. On Nov. 19, 1927, peri-arterial sympathectomy was performed on the left brachial artery.

When seen in May, 1928, he stated that he had been completely free from symptoms, and was at work.

This case shows that peri-arterial sympathectomy will quickly relieve the symptoms of Raynaud's disease.

Case 2.—A married woman, age 35, had suffered from syncope of her hands and feet for ten years. Since childhood she had noticed that her hands became blue on the least exposure to cold, but for ten years the typical spasms of Raynaud's disease had occurred spontaneously. In spite of courses of drugs, massage, endocrine therapy, and artificial sunlight, the disease had progressed. The artificial sunlight had intensified a tendency to scleroderma, and general desquamation had occurred for two months after this treatment had ceased.

She came under my notice in January, 1927. The fingers of both hands as far as the nail-beds were shrivelled and black. Many of the toes were similarly affected. Whilst under observation typical spasms of Raynaud's disease were seen on many occasions. As gangrene had progressed further in the right hand than the left, and as she complained more about the right hand than any other limb, peri-arterial sympathectomy was performed on the right brachial artery on Jan. 24, 1927. The artery was very small, about one-sixth the size of a normal vessel. Four days after the operation

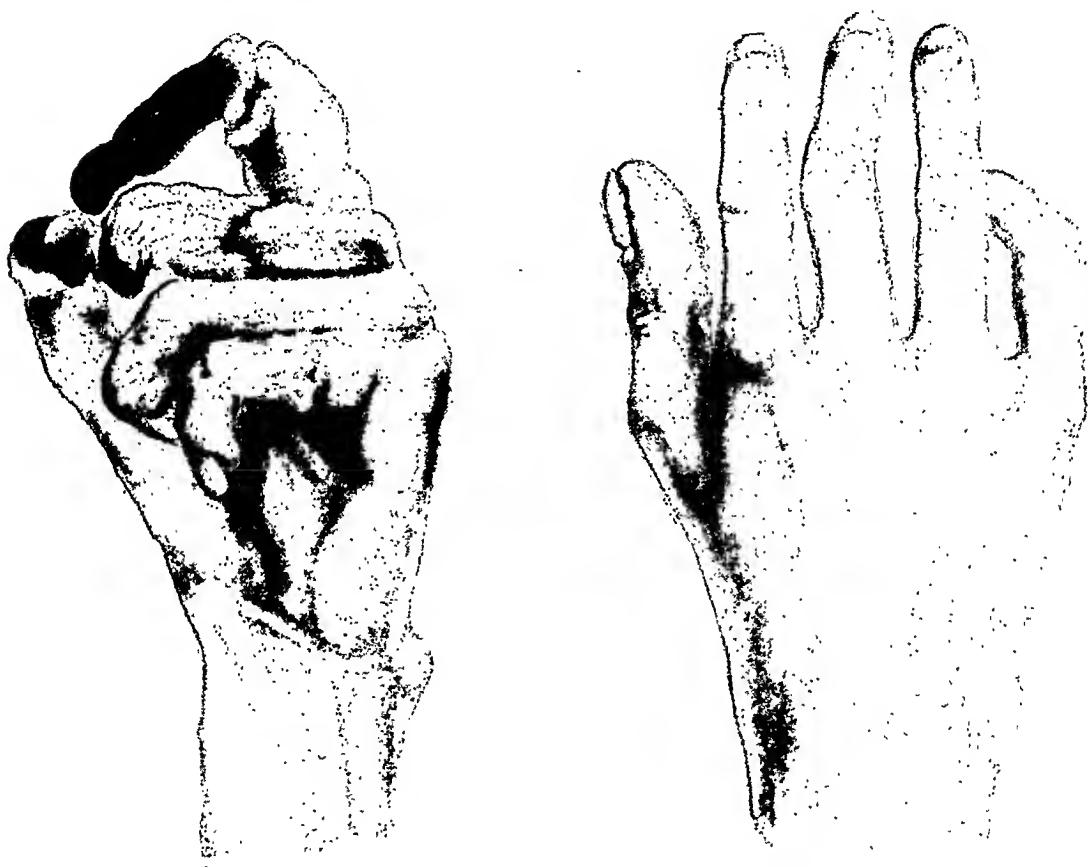


FIG. 176.—Coloured drawing from life fifteen months after peri-arterial sympathectomy on the right brachial artery. The disease has advanced apace on the unoperated side (left), whilst on the side upon which the sympathectomy was performed (right) not only has the disease been arrested, but also there is very marked nutritional improvement.

it was noted that during a paroxysm of the disease the left hand was alone involved.

Six weeks later moist gangrene commenced in the right foot and began to spread rapidly towards the middle of the calf. This was treated by amputation in the lower third of the thigh, preceded by a sympathectomy on the femoral artery. She made a slow recovery and refused any further operative treatment.

I have kept the patient under intermittent observation. The disease has progressed very extensively in the left hand and the left foot, whereas the right hand, which originally presented the most advanced signs and of which the patient most complained, now presents a comparatively normal appearance and is the only useful member. The illustration (*Fig. 176*) was drawn from life fifteen months after peri-arterial sympathectomy had been performed on the right brachial artery.

This case is unique, for we have in the unoperated side a control. Upon the operated side we find that the disease has not only been arrested, but also, after an interval of fifteen months, shows very considerable improvement, whilst on the unoperated side it has advanced apace.

The conclusions are : (1) That peri-arterial sympathectomy performed on the main artery supplying a limb will quickly alleviate the symptoms of Raynaud's disease in that limb : (2) That there is a certain amount of evidence to justify the hope that this relief is permanent.

REVIEWS AND NOTICES OF BOOKS.

Clinical Researches in Acute Abdominal Disease. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S., Senior Surgeon to Out-patients, St. Mary's Hospital, London. Second edition. Pp. 214 + xvi, with 44 illustrations. 1927. London: Humphrey Milford. 10s. 6d. net.

THE second edition of this book contains three new chapters—one on extravasation of bile, one on acute ascending parenchymatous enteritis causing paralytic ileus, and one on shock and collapse, with special reference to acute abdominal disease.

In the chapter on extravasation of bile, cases that have come under the personal care of the author are quoted. The condition is divided into retroperitoneal and intraperitoneal extravasations, the latter of which are the commoner. The different views as to the state of affairs that allows of the extravasation are shortly described—that of filtration and that of the presence of a minute undiscovered perforation of the bile-duets or gall-bladder. On the whole the latter is probably the cause in most cases, although in many of those mentioned no perforation either at operation or at autopsy could be discovered. The clinical symptoms are variable, but one especially mentioned by the author is the remission of symptoms after the first acute onset. This may be due to the temporary stoppage of leakage, by the omentum, of bile which is secreted under very low pressure. At present our knowledge of the early signs and symptoms of this condition is not very accurate, and this is borne out by the fact that the mortality in the author's cases amounted to no less than 60 per cent.

The longest chapter in the book is a new one on "Shock and Collapse". This was given by the author as a Hunterian Lecture at the Royal College of Surgeons. He points out the still existing difference of opinion both as to the definition and essential nature of the condition of 'shock'. The great difficulty seems to be the absence of a suitable definition of the word 'shock', especially from the purely clinical point of view.

The clinical symptoms of shock are grouped into three classes: (1) Those due to circulatory failure: pallor, superficial cold, mental dullness, increased pulse-rate, diminished pulse-volume, lowered blood-pressure. (2) Those due to depressed metabolism: subnormal temperature, diminished secretions and excretions. (3) Those due to sympathetic stimulation: sweating with consequent thirst, dilated pupils. The author says that these symptoms may be dissociated in varying degree, but in demonstrable shock there is nearly always either subnormal temperature or a lowering of blood-pressure, and usually both. To this we would add an increased pulse-rate, as he does in his first group of clinical symptoms.

Several authors and cases are quoted to prove that a patient with a slow pulse may be suffering from shock, but these cases seem to be exceptional. In the same way authors are quoted to prove that a low blood-pressure is not an essential accompaniment of the clinical picture of shock. For instance, "It is probable that shock may be present before the blood-pressure falls, though it is difficult to recognize its presence except that the patient may look bad". Again, "Although most shocked patients have a low blood-pressure and rapid pulse, there are some notable exceptions". This latter the author considers to be the stage of latent shock.

Perforations of the duodenum and stomach are specially mentioned as causing the appearance of shock, and Sir Berkeley Moynihan is quoted to substantiate this statement, though this authority denies the existence of 'shock' in the true surgical sense. The author agrees that difference of opinion depends to a certain extent on the definition of the word 'shock'. Either that definition is imperfect or the

proof of its correctness should be specially demonstrated by careful and detailed data. We are inclined to think that the former is the cause of this difference of opinion. We agree with Sir Berkeley Moynihan that in the early stages of perforation of duodenal ulcer the absence of a rapid pulse, lowered blood-pressure, and diminished blood-volume in the vast majority of cases should preclude the use of the word 'shock', a word which to the majority of practitioners calls up a picture of a rapid pulse and a lowered blood-pressure. Cases are mentioned suffering from what the author calls 'shock' who have a slow pulse and a normal blood-pressure, but we think these are exceptions to the general rule. He says that all authorities agree that shock is present in cases of acute pancreatitis—that is, the pulse is rapid from the onset of the symptoms—but quotes one case in which the pulse was slow and which was operated upon as a likely case of perforation of a duodenal ulcer. This again was an exception to the general rule that shock is usually accompanied by a rapid pulse. Patients suffering from a perforation of a duodenal ulcer do not in the early stages show the essential signs of what we consider to be usually accepted as those of shock—that is, a rapid pulse, a lowered blood-pressure, and a diminished blood-volume.

The picture of a patient suffering from shock with a rapid pulse and a lowered blood-pressure is so firmly impressed upon the mind of the average practitioner, that we think it would be wiser to use some other expression to denote the condition of a patient in the early stages of perforation of a duodenal ulcer. Such an expression as 'prostration' might be adopted, as was suggested by Sir Berkeley Moynihan in his latest article in the *Practitioner*, January, 1928, on perforations of gastric and duodenal ulcers. He there says shock in the surgical sense is absent. The word 'shock', used in its strict sense, means a condition in which increased pulse-rate, diminished blood-pressure, and reduced blood-volume are present. None of these is found in the first hour or two after a perforation has occurred. Shock comes on later—when peritonitis has developed. This, we think, represents the true state of affairs in the very large majority of cases, and we would suggest that perhaps the word 'prostration' more clearly defines the condition of a patient in the early stages of a perforation.

The book is one which we can strongly recommend to those who are interested in the more scientific side of that very important subject, the acute abdomen.

Die Leitungsbahnen des Schmerzgefühls und die chirurgische Behandlung der Schmerzzustände. By Professor Dr. O. FOERSTER, Breslau. Royal 8vo. Pp. 360, with 104 illustrations. 1927. Berlin and Vienna: Urban and Schwarzenberg. Sewed, M. 19.50; bound, M. 21.90.

A book on pain from the pen of Professor O. Foerster is naturally an event in current medical literature, and his new book *The Pathways of Pain and the Surgical Treatment of the Algeias* is in many respects the most important work on the subject which has yet appeared.

It is divided into two sections, in the first of which the anatomical paths of pain, from the receptors through the peripheral nerves into the posterior roots and posterior horns, are followed step by step in considerable detail, and the applied physiology of the transmission of pain is discussed. In six further chapters the pathways are traced through the cord and brain-stem to the final end-stations in the thalamus and cortex cerebri. In the second division of the book pain is discussed in its clinical aspects in so far as it is occasioned by affections of the different segments of the conducting mechanism.

Foerster's name has latterly become associated with the idea that the vessels are media for pain conduction. Peri-arterial sympathectomy has taught us that the great vessels are sensitive in a high degree, though apparently not all to an equal extent. Foerster mentions that L. R. Müller denies pain fibres to the cerebral vessels, but Foerster has found ligature of these vessels frequently productive of pain, as for instance during local excision of cortex. He has also found the injection of an

angiodilator (papaverine), or direct injection of calcium bromide into the carotid, an instantaneous cure for migraine. He mentions a most interesting case of rupture of the lower roots of the brachial plexus (C 7, T 1) distal to the entrance of the rami communicantes. Although the skin was anæsthetic from the third to the fifth finger, deep pain was not abolished. He exposed a digital nerve and stimulated it with the faradic current without producing any pain. He then freed the corresponding digital artery, and electrical stimulation of that immediately produced severe pain. Foerster restates his views on the sensibility of the dura in view of Müller's criticism, and asserts that the dura of the cranial base and of the tentorium is exquisitely sensitive to cutting and sometimes even to stroking, but that it becomes less and less sensitive towards the vertex. The dura in the region of the main trunks of the middle meningeal arteries is also very tender. Experience varies on these points, and indeed the author himself admits that there is no constancy in these findings from one case to the next. In contrast to this he has not been able to produce pain from stimulation of the pia mater even when it was inflamed. The spinal theca he finds less sensitive to pain than the dura mater.

When we come to the posterior roots, we find a number of most helpful photographs of patients with the areas of sensory defect after root-section most carefully plotted out, and these in themselves are a most valuable contribution. Some of the delimitations have been obtained by the reverse method—electrical stimulation of the cut peripheral end of a root, for this is followed by an immediate outspoken vasodilatation of the skin, thus enabling one to identify accurately the particular root cut and to satisfy oneself that a sufficiently wide area is being denervated.

On the vexed question as to whether or not the anterior roots conduct sensation or any part of sensation, Foerster makes some interesting observations. He regards the anterior roots as an auxiliary sensory pathway ('accessorische Hilfsbahn'). In a diagram illustrating the afferent fibres of the anterior root he draws a branch of a posterior root axon and a sympathetic fibre entering the radix anterior. He has produced severe pain following stimulation of the central end of a cut anterior root. It varies from one nerve to another. In one case he cut the dorsal roots 6 to 10, and whilst the 8th anterior root on stimulation produced maddening pain, in a parallel case a scarcely painful unpleasant feeling resulted from a similar stimulation. In the first case, after the cutting of the posterior roots, deep sensibility had been very little altered, in the second profoundly changed. It is well known that division of the sensory root of the Gasserian ganglion does not abolish deep sensibility in the face, but Foerster has found that by dividing the hypoglossal as well, pressure pain was abolished in half the tongue. He has also found the central end of the facial nerve to produce lively pain on stimulation. As for the existence of an extra-radicular pain pathway in the sympathetic, he mentions a case in which he extirpated the Gasserian ganglion, cut the cervical sympathetic, and performed a peri-arterial sympathectomy on all the carotid arteries. The result was that pressure sense and pressure pain were diminished but not abolished. Foerster goes on to discuss the pathway of deep sensibility in the treatment of gastric crisis. It is interesting to find him producing clinical evidence of pain from lesions of the posterior column, a fact with which all will be familiar who have read the account which Brown-Séquard gave of his own famous animal experiments.

A most excellent section is the one on posterior horn pain, containing several observations on pain arising from central gliomatosis. He comments on the frequency of spontaneous pain in intramedullary tumours, and says that in all the cases of this kind which he has observed, pain in the dermatomes corresponding to the site of the tumour was the first symptom. He refers particularly to a cylindrical central glioma which began in the middle of the thoracic cord and spread upwards and downwards from there. Shortly before death, analgesia and thermæsthesia extended on both sides from the 2nd cervical to the 4th lumbar segments. Its gradual spread upwards and downwards was traceable clinically by the development of spontaneous pain in successive dermatomes, to be followed by analgesia. He has observed the same march in syringobulbia, and has watched pain spread from the trigeminal field to C 2-4.

He records observations confirming the fact that pain and temperature sense may be dissociated in the posterior horns no less than in the spinothalamic tract, and the temperature sense itself may be dissociated into hot and cold elements of different distributions. We can allow ourselves only one more quotation. He now states categorically that the pain fibres in the crossed spinothalamic tract need only one segment to reach their position, and not the three or four which it is the common usage to allow them. With this statement the reviewer finds himself in complete agreement. Foerster mentions, for example, a case of bilateral section of this tract at the upper end of the 5th thoracic segment, and all dermatomes from T 6 to S 3 were analgesic and thermanæsthetic. There can be no doubt in our mind that the depth of the cut is the most important factor in determining the level of sensory defect, and Foerster appears to be correct in assigning the most superficial position to the fibres from the lowest levels, whilst those from higher levels are much deeper. In his schema of the sensory and motor pathways in cross-section he shows lamination of the main sensory pathways (which we can accept in great part), but he also laminates the pyramidal tract in the cord, and for this we can find no justification.

Foerster appears to have antedated Spiller and Frazier in section of the spinothalamic tract, for he mentions performing this operation in 1912 for tabetic pain. He states (pp. 108 and 109) that the first experimental knowledge on the spinothalamic tract as a pathway for pain was obtained by himself by accident. In injecting a case of intercostal neuralgia in the 6th intervertebral foramen the needle point went in too deep and entered the cord, the patient complaining of terrible pain in the opposite leg, and he mentions that severe pain may follow any handling of this tract.

So much for the general arrangement; it is clear that we have here a fuller account of the pain-conducting system than exists in any monograph heretofore. It is a book which everyone interested in pain and its treatment should have and reflect on. We have quoted freely from the monograph, since there is so much clinical fascination to be found in it, for the general surgeon no less than for the neurologist.

It is clear that Professor Foerster has allowed no opportunity to pass for enriching his own experience, and we are grateful to him for the knowledge which he has passed on. It seems ungracious to complain, but why is it that even the most important of Continental books are allowed to appear without an index? The volume is illustrated by a great number of photographs and diagrams, all of which have been most painstakingly prepared.

"*L'Anæsthésie Régionale.*" By VICTOR PAUCHET, P. SOURDAT, G. LABAT, and R. DE BUTLER D'ORMONT. Fourth edition. Royal 8vo. Pp. 296, with 328 illustrations. 1927. Paris: Gaston Doin et Cie. Fr. 55.

MANY books have appeared in recent years dealing with the subject of regional anæsthesia, but this one, which has now reached a fourth edition, still retains its place in the forefront. To read any book on this subject is to realize how easy it is to produce a perfect anæsthesia; it is not until one tries it that the difficulties and disappointments present themselves. Dr. Pauchet sums up the position very well when he says: "Do not condemn it untried. Its users are constantly making advances: actually there is not one student of medicine who can ignore this method of anæsthesia. There is not a surgeon who can do without it in his practice. Those who condemn it, do not know it. To know it, one must learn, and this book will teach the methods."

This last claim is well founded. No trouble is spared in the text to make every step as clear as possible, and every procedure is amply illustrated by the very numerous plates. The method of arrangement is good: an account is given in the beginning of the method of anæsthetizing any nerve-trunk in the body, and in the later part of the book the operations which can be performed on the different parts of the body are considered in turn. In each case the nerves which must be dealt with are enumerated, and one is referred back to the earlier pages for details of the

technique. Unfortunately in several places the cross-references are omitted, an error in the editing which, although trivial, is rather irritating.

The book has been well produced, in particular the illustrations, which are both numerous and instructive. As a text-book for the beginner, or as a book of reference for the more expert, we can confidently recommend this work.

Local Anæsthesia. By G. DE TAKATS, M.D., M.S., Assistant Professor of Surgery, Northwestern University Medical School, Chicago. With a Foreword by ALLEN B. KANAVEL, A.B., M.D., D.Sc., Professor of Surgery, Northwestern University Medical School. Medium Svo. Pp. 221, illustrated. 1928. London: W. B. Saunders Co. 18s. net.

THE use of local anæsthesia would seem to be extending in America. Certainly local methods often present advantages. The author of this book graduated in local anæsthesia in Budapest and has continued his experience amongst the American people; he can therefore speak with some authority. His book is a summary of present-day practice. It is not too long, and is very clear in its directions. He is dogmatic in his statements, but they are always carefully considered. We welcome particularly a book on this subject on which the author is not over-enthusiastic. De Takats does not recommend paravertebral or posterior splanchnic anæsthesia for abdominal work, believing them to be unsafe. He has also seen accidents from deep cervical block anæsthesia. For thyroidectomy he substitutes a combination of superficial cervical block with injection round the upper poles of the gland. Though individual surgeons will not agree with all the statements made in the book, it can be recommended as a concise reliable guide to those inexperienced in this method of anæsthesia. In the last line of page 174 'cervical' should read 'dorsal'.

Bone Sarcoma. By H. KOLODNY, Ph.D., M.D., Department of Surgery, State University of Iowa. Royal Svo. Pp. 214, with 104 illustrations and 41 plates. 1927. Chicago: Surgical Publishing Company.

THIS monograph is based almost entirely upon the material from the Registry of Bone Sarcoma of the American College of Surgeons. In this Registry, which was started in 1920, no less than 700 cases have been tabulated. The author gives reasons for rejecting the parasitic origin of sarcoma, and for considering it to be due to an alteration in growth restraint after trauma.

It was inevitable that any new review of a complex subject such as this should lead to a proposal for fresh classification, and equally inevitable that much of such new classification would prove very unacceptable to other workers. In the first place, all the ordinary malignant sarcomas the tissue of which arises from bone-forming elements are termed osteogenic sarcoma. Thus the time-honoured distinction of spindle-cell or round-cell, as well as that of periosteal or endosteal, are abolished, and for this we think that full and convincing reasons are given. It is the same as the abolition of the distinction between periostitis and osteomyelitis in acute affections of the bone. Periosteal fibrosarcoma is admitted to be a real entity, and is a tumour of low malignancy of extraperiosteal origin. The greatest difficulty to the English reader is presented by what is termed Ewing's tumour or Ewing's sarcoma. This is described as a malignant endothelial growth occurring in the mid-shaft of the pipe bones and in the small bones. It arises in young patients and is usually multiple. It closely resembles chronic osteomyelitis in its X-ray appearance. In addition to the usual clinical evidences of pain following trauma, the cases present repeated febrile attacks, and if it were not for the absence of leucocytosis the condition might easily be confounded with leukaemia. If time should confirm the reality of this as a separate entity it is most sincerely to be hoped that a suitable descriptive name may be found for it. Myeloma is a name reserved for cases of multiple bone-marrow tumours associated with Bence-Jones albumosuria, and the tumour which English writers usually call myeloma is called a giant-cell tumour.

The largest section of the book is devoted to the description of osteogenic

sarcoma, and 41 pages are given to an account of the multiform histology of this disease. The microphotographs, and indeed all the illustrations, are models of clearness and of excellent reproduction. Two little points of criticism we would suggest, however—that the figures should be numbered consecutively throughout, instead of being separately numbered as plates or figures, and also that the degree of magnification of the microphotographs should be indicated. Much statistical and clinical information of great interest is given. It is reckoned that about 1 in every 100,000 of the population is affected with bone sarcoma. Males to females are as 4 to 3. The age of maximum frequency in both sexes is 20. The proportion of cases in the arm and leg is as 2 to 11; the right side to the left side as 5 to 4. The order of frequency in individual bones is as follows: femur, tibia, humerus, pelvis, fibula, shoulder girdle, ulna, hands, feet, ribs, skull, jaws, and vertebrae. There has been no case registered in the radius or lower end of the tibia. About 5 per cent of all bone sarcomata occur in Paget's disease. Pain precedes tumour formation by an interval of days, weeks, or months; it is worse at night and is relieved by radiation. Trauma, which is usually a trivial sprain or contusion, precedes symptoms by about one month. Radiation has such a definite effect in allaying pain and in making the X-ray picture more sharply defined that it is regarded as an important diagnostic test. Biopsy should never be employed as a diagnostic test except under the precaution of a double elastic tourniquet, and then only as a preliminary to amputation if the section should reveal a malignant growth. The author suggests that the best routine treatment should be: (1) Radiation followed by splinting and recumbency; (2) Amputation; and (3) Prolonged prophylactic radiation of the chest. He considers that the cellular structure is of very little value in prognosis.

The last 45 pages are devoted to an account of the giant-celled tumour, which is of benign nature and which can usually be treated by conservative methods.

The wealth of illustrations and of clinical information make this monograph one of great value for reading and for reference.

Neoplastic Diseases: A Treatise on Tumours. By JAMES EWING, A.M., M.D., Sc.D., Professor of Pathology at Cornell University Medical College, N.Y.; Pathologist to the Memorial Hospital. Third edition. Royal 8vo. Pp. 1127, with 546 illustrations. 1928. Philadelphia and London: W. B. Saunders Co. Ltd. 63s. net.

THIS is the third edition of a book which requires no introduction; for the student, and more particularly the practising pathologist, already knows it as a valuable book of reference, and an amazingly complete guide to the literature of tumours.

When comparing this with the preceding editions it is plain that the book has been very carefully revised; and there have been so many changes, great and small, in the framing of individual paragraphs, that one might almost say that the book has been re-written. The illustrations, which are excellent, have also been gone over, and many new ones have been added.

Innocent as well as malignant tumours are described, though the object of the work still stands out—to be a study and record of cancer. The scope of the work is widened still further by the inclusion of certain conditions (especially in the section on bones) which lie in the borderland between inflammation and true tumour formation, and diseases of the endocrine organs which are more functional errors than neoplasms.

The sections dealing with the theories of the nature of cancer, experimental cancer research, sarcomas and endotheliomas of bone, and brain tumours, have been almost completely re-written and brought up to date. Many new facts and illustrations appear in the descriptions of liposarcoma, chordoma, carcinoma of the thyroid, and pineal gland tumours.

Although the subject is dealt with primarily with a view to expounding the natural history and the structure of neoplasms, yet frequent reference is made to the clinical aspects of tumour formation, and this is of the utmost value to the clinician as well as to the pathologist.

The main criticism we would offer is in regard to nomenclature. When reading certain chapters—for instance, those on the tumours of bone and on renal tumours

—it is not possible for a person brought up on English books to follow Dr. Ewing's descriptions until he has 'translated' the terms into another language. This problem of nomenclature in pathology is one which demands the serious consideration of investigators and teachers, so that the science of pathology may be international, and that new work may be appreciated by workers in the same field all over the world.

Allgemeine und specielle chirurgische Operationslehre. By Professor MARTIN KIRSCHNER and Professor ALFRED SCHUBERT, Königsberg. Royal 8vo. Vol. I: General Surgery. Pp. 648, with 709 illustrations, for the most part in colour. 1927. Berlin: Julius Springer. M. 114; bound, M. 120.

THERE can be no doubt as to the importance of this new German work. At the first glance one realizes that it represents an effort to attain the very best that is possible in text and figures, and further detailed perusal serves to confirm this first impression. Although large and comprehensive, the book is the product of only two authors, and in this respect it compares very favourably with those systematic treatises which emanate from a great number of specialists. There is a uniformity and proportion in the various chapters which is lacking in the composite text-book.

The first chapters of over one hundred pages are concerned with general principles of the operation room, the investigation of the patient before operation, and the methods of placing the patient and of wound suture.

The second section is devoted to the abolition of pain by the various methods of anæsthesia. In regard to the descriptions of inhalation anæsthesia, we would remark that the usual intratracheal methods are not given due prominence, although full mention is made of the use of Kuhn's and of Hahn's laryngeal tubes. All methods of local anæsthesia, whether by infiltration or by nerve blocking, are fully described, and the beauty and accuracy of the drawings make this section very valuable. Then follow chapters on aseptic technique and the operative treatment of local infection; the arrest of hæmorrhage by forceps, ligature, and tourniquet, and the treatment of the effects of loss of blood by saline and blood transfusion. The fifth chapter, on operations on the skin and subcutaneous tissues, is introduced by some most useful and beautifully drawn figures of the lines of tissue tension in the skin over all parts of the body. There follows an excellent and fully illustrated description of various plastic operations, including the use of reversible tube-flaps and methods of epithelializing cavities and sinuses. The sixth chapter gives a complete account of operations for tendon transplantation, including the repair of tendons by free fascia flaps. The eighth chapter, on affections of the blood-vessels, mentions a method of treating cavernous angiomas by implantation of bits of metallic magnesium which is new to us. Operative proceedings on varicose veins are given in great detail; injection methods are commended, but only in conjunction with ligature of the internal saphenous vein; no fewer than seven other operations are described and illustrated, and this is an example of the value of this work for its completeness as a book of reference. The next chapter is on the surgery of the peripheral nerves, and is very complete. It includes all the classical procedures for nerve-bridging, together with several new suggestions, e.g., that for dealing with an amputation neuroma by implanting the freshened nerve-end into the side of the nerve-trunk. The figure (520) of peri-arterial sympathectomy is a good example of the combination of anatomical accuracy and artistic merit which is a great feature of this work. The presentation of essential parts in colour whilst the main drawing is in black and white gives a very good effect. The last three chapters deal with operations on bones, joints, and amputations. They are admirable in the clearness of description and in the wealth of illustration. Elaborate methods of bone suture by wire receive, we think, attention which they do not merit. Methods of comminution osteotomy are described which are unusual, but their value is obvious. In describing arthroplasty general principles are given in connection with the knee and elbow, but we should like to have had full details about the hip-joint.

The excellence of this first part of the work makes us eager to receive the second volume dealing with operations upon special organs.

Emergency Surgery. By GEORGE DE TARNOWSKY, M.D., F.A.C.S., D.S.M., Col. M.C., Professor of Clinical Surgery in the Loyola University Medical School. Medium 8vo. Pp. 718 + xvi, with 324 illustrations. 1926. Philadelphia and New York: Lea & Febiger. \$7.50.

THE title of this book may prove a trifle misleading to those who look to find an account of the general emergencies in surgery. It deals only with injuries and wounds, the result of accidents. The book is intended primarily for the instruction of industrial surgeons, and is written by one who has had an extensive experience in all sorts of injuries, including battle casualties and the accidents of civil life.

A great deal of interest is now being taken in industrial medicine, and employers of labour and big corporations are finding that "the medical care and attention given to an injured worker determine to a large degree the time of his return to productive employment". So gradually institutions for the care of workers, and literature on the subject, are beginning to spring up, and Mr. Tarnowsky has produced a long, detailed account of what to do in every kind and grade of wound from the crown of the head to the sole of the foot. One cannot help being reminded of the type of book known in the past as "*Wund Artzney*" in which the mediæval students were instructed how to deal with injuries of the head or stab wounds, and every variety of contusion and dislocation. This book is in fact an up-to-date complete instructor in all that pertains to wounds and accidents, and it may prove of inestimable value to many who are called upon to treat accidents. There is one chapter whose importance is rather difficult to assess, namely, that dealing with the medico-legal aspects of surgery; though the laws of the United States and Great Britain are not identical, still this chapter seems worth reading.

The Tongue and its Diseases. By DUNCAN C. L. FITZWILLIAMS, C.M.G., M.D., Ch.M., F.R.C.S., Surgeon and Lecturer on Surgery to St. Mary's Hospital. Demy 8vo. Pp. 505, illustrated. 1927. London: Humphrey Milford. 36s. net.

THIS volume sets forth what the author believes to be the modern surgery of the tongue, based upon a wealth of clinical and pathological material and an astounding amount of information culled from English and foreign literature. Every section of the book contains detailed references to and abstracts from the published accounts of diseases of the tongue, and a very full bibliography is appended to every chapter. We must confess that although these references are of interest, yet the greater number of them cannot be of much value except to those who, when meeting with a rarity, may be anxious to discover where and when it has been met before. All such must be deeply indebted to Mr. Fitzwilliams for his wide review of the literature.

The early chapters deal with the anatomy and development of the tongue, wounds, and acute and chronic inflammation. Then follow four chapters on the salivary glands, ranula, dermoids, and thyroglossal tumours and cysts, which are, in our opinion, the best in the book. They are well illustrated, and deal with these somewhat confusing subjects in a clear and practical manner.

The rest of the work is devoted almost entirely to cancer of the tongue. The author states in the preface that "the chief lesson of this book is that cancer of the tongue . . . is just as curable in its early stages as is cancer elsewhere". Although it is stated (no doubt correctly) that in early diagnosis and treatment lies the only hope for better results in the surgery of cancer of the tongue, yet we cannot find any evidence produced by the author from which we may learn the chief lesson of the book. The description of operations is followed by figures from many sources, which show merely that a large percentage of patients present themselves for treatment when their disease has become inoperable; and that of those operated upon, only a small percentage survive for three years.

In dealing with the two-stage operation, the advantages of carrying out the removal of the tongue first and the glands later are dwelt upon, without reference to the drawbacks of this procedure. Diathermy is regarded with disfavour, and it appears to us that insufficient stress has been laid upon treatment by radium, using modern technique. No mention is made of the adjuvants to anæsthesia which tend to reduce the number of inoperable cases.

By its nature and its cost the book is rather a work of reference for the specialist than a text-book for the student. Under the circumstances the illustrations should be improved to conform to the standard of the rest of the work.

Curietherapie: Basi Fisiche e Biologiche: Applicazioni. By CARLO PEDRAZZI. Demy 8vo. Pp. 130, illustrated. 1927. Bologna: Licinio Cappelli. L. 35.

THIS monograph deals with the physics, the biological effects, and the applications of irradiation. It is based mainly upon the technique and general work of the Paris Radium Institute, and gives a readable and reliable account of radio-activity, radio-sensibility, and the principles of treatment. Rather more than half the volume is devoted to accounts of regional treatment as practised by Regaud and already well known. Even the illustrative photographs are mainly borrowed from French sources. There is a bibliography of ten pages.

Le Fratture del Gomito. By F. ROSSI. With a Preface by Professor BALDO ROSSI. Medium 8vo. Pp. 312, with 246 illustrations. 1927. Milan: Coop. Grafica degli Operti.

THIS monograph, based on 503 cases of fractures about the elbow-joint, has as its object to furnish evidence from Professor Baldo Rossi's clinic at the Ospedale Maggiore that results as good as any to be attained by operative methods may be achieved without the knife, by careful radiography, skilful reduction, and unremitting attention to after-treatment.

A great many cases of supracondylar fracture are illustrated by serial radiographs, and well sustain the claims made for non-operative measures, whether treated by the acute-angle method or by weight extension, as conditions necessitate. The clinic is not without its difficulties and problems, but the number of cases of ankylosis, false joint, and deformity appears to be small. In fractures of the external condyle the author states that it is only rarely necessary to resort to operation if repeated manipulations with X-ray control be perseveringly adopted.

Of eleven cases of fracture of the internal epicondyle, nine were treated without operation and with entire success; and much the same may be said of the cases of fracture of the whole internal condyle, though the difficulty of preventing ultimate deformity is recognized. T-shaped fractures can generally be manipulated into place and maintained by a posterior plaster splint, but extension may be necessary. Separation, partial or complete, of the epiphyses, is treated by immobilization for as short a time as possible, followed by the most careful passive movements.

The latter part of the book deals with fractures of the upper ends of the radius and ulna; in every case details of the manipulative reduction, duration of treatment, and end-results are given, and cases illustrated.

As a work of reference, both as to methods of other clinics, and particularly as to that of Professor Rossi, the book is extremely valuable, and a notable propaganda for manipulative technique.

Die chirurgische Behandlung der Gehirntumoren: eine klinische Studie. By HERBERT OLIVERONA. Royal 8vo. Pp. 344, with 228 illustrations. 1927. Berlin: Julius Springer. M. 27.

IT may be said of neurological surgery that the monographs which it has stimulated its devotees to write are amongst the most distinguished contributions which have seen the light in our time. Most have been confined to some particular pathological entity, but Dr. Herbert Oliverona, of Stockholm, treats of intracranial tumours as a whole, and once more we have a book of unusual merit and one from which the surgeon working in this field can derive the greatest information. The book is essentially a personal record of four years' intensive experience. It illustrates well the fact that any man who takes up a special line for study will accumulate material that no one believed to exist. The point is well exemplified in the

Stockholm statistics. In the ten-year period 1912-21 only 31 cases of verified intracranial tumours were operated upon at the Serafimer-Krankenhaus, whilst in the four years 1922-26, during which time Olivecrona had full charge of a neurological service, there were 105 cases of tumours or tumour-suspects, of which 75 were verified (41 gliomas, 11 meningiomas, 12 neurinomas, 2 tuberculomas, and so forth). In a number of detailed tables the reader will find interesting analyses, but above all he will be interested and instructed by the admirable detailed descriptions of each and every one of the cases. The same plan is followed for all patients; first a short general history, then general pressure symptoms and localizing symptoms and signs, followed by the X-ray report and a complete account of the surgical treatment.

The clinical histories and the neurological signs are briefly and concisely given; in no case is there a long dissertation on this side of the problem, the one which has most enchanted the English mind in the past. And yet one is told enough to appreciate the case fully and to enjoy the problem presented. Olivecrona has made use of the common surgical aids to diagnosis. Radiological help (if one may be permitted to enlist as 'surgical' a neutral art) he has had from Dr. Lysholm, and those familiar only with English and American technique will be delighted with the revelation of yet another superb technician, strengthening Stockholm's claim to pre-eminence in the X-ray world.

The author used exploratory ventricular puncture in 13 cases, ventriculography in 14, and encephalography (lumbar air injection) in 4. The last produced the severest reactions, but the deaths, of which there were 2, followed ventricular injection—one a case of cerebral metastatic carcinoma, the second a deep glioma.

Olivecrona has made direct attacks upon all his tumours and has extirpated as many as possible. His mortality for decompression for gliomas is high: 7 deaths out of 9 cases of subtemporal, and 1 death in 2 suboccipital decompressions. Of 10 exposures and decompressions 5 died, whilst of 8 extirpations or treatment of cysts only 2 died. Thus the mortality varied inversely with the amount that was done, from which one infers that it was not the operation itself which brought a high mortality in its train, but the advanced nature of the case. The more benign cystic gliomas without underspread reactionary oedema are very favourable; others will succumb with the slightest interference, especially perhaps if the decompression cannot be placed accurately over the tumour. Oedema is unquestionably the bugbear of the glioma group, and is more important than hæmorrhage into the tumour, not because the latter does not occur but because it is certainly rarer. These arguments are pointed by an analysis of the general clinical condition of the patient. At the time of operation 7 were in coma, and 6 of them died; 10 had advanced generalized pressure, and 6 died; 11 showed only slight pressure changes, and of these only 3 died. Objection may be raised to the fact that the gliomas have not been well differentiated histologically, and a retrospect over each case from this viewpoint would have enhanced the already great importance of the book. Olivecrona's relatively large group of acoustic neuromas he owes to early recognition of the syndrome by his colleagues and himself: 3 of the 9 examples had no sign of choked disc. In general, 'choke' was present in 86 per cent of his verified tumour cases.

The author gives a full account of his operative methods, which do not differ in any essential point from those in general use amongst specialists to-day; but those eager to learn will find things of interest, and often something to *admire*, in the complete account which is given of the treatment of each one of the cases. Olivecrona uses local anaesthesia for choice, and does not hesitate to break off the operation and continue it in two or three days (not the classical two or three weeks which was the custom some years ago) if the patient is well, or more particularly if he is going downhill and something useful remains to be done. We are learning that a life may sometimes be saved by a bold second intervention, however recently an operation may have been performed. He has unquestionably risked some cases in which little or nothing could be hoped for—his comatose glioma patients and the angiomas are cases in point—but the critic's task is easier than the surgeon's.

stop before the patient feels any pain in the loin : otherwise, the pelvic muscle goes into spasm or shows irregular and violent contractions, in either case vitiating the findings ; and, finally, that they withdraw the catheter after filling the pelvis. They point out that the catheter, which must be opaque to the X rays, should only just enter the pelvis : if pushed too far it may engage in the uppermost calix, which a very small amount of fluid will suffice to distend and so start these undesirable contractions. They also emphasize the importance of waiting until the observers' eyes are thoroughly accustomed to the dim light.

The only part of the apparatus which is not simple and inexpensive is the camera which they use in conjunction with the screening, so that, at any given moment, a plate may be exposed and a permanent record obtained ; although this was obviously necessary for the purpose of the present work, we imagine that a tracing of the condition found would answer in ordinary routine work.

If, as sometimes happens, the catheter is arrested at some point in its passage up the ureter, the patient is taken to the X-ray room and opaque fluid is injected to ascertain the cause of the obstruction : by this means they found that the arrest is due to a kinking of the ureter and can be overcome by pushing up the kidney ; in over a thousand cases they never found a single case of stricture of the ureter ; this seems to us to be an observation of considerable importance in view of the great difference of opinion amongst urologists as to the frequency of this condition.

They state that if the catheter is left in the ureter with its end anywhere short of the pyelo-ureteric junction, the fluid will, as a rule, flow back along the side of the catheter, and that it is only in cases of atonic ureter that a pyelogram can be obtained under such conditions : this statement will come as a surprise to many urologists, and would seem to make the X-ray diagnosis of ureter fissus more difficult than ever.

The authors describe a 'ureteral bulb' which they compare with the duodenal cap : to this they ascribe a good deal of importance, and they consider that the X-ray appearances seen here are a criterion of the motility of the pelvic musculature. It is situated at the junction of the pelvis with the ureter, and normally a drop can be seen to form here every three to seven seconds ; after reading their account of the formation and behaviour of this drop, one is irresistibly reminded of the drop that forms at the outer-world end of a ureteral catheter, and one wonders whether too much importance is not attached to the ureteral bulb ; however, they attach great significance to the careful observation of this bulb, which they think is the visible expression of a sphincter at that level, and they give in detail the modifications undergone by this bulb in disease : in fact, they have observed both retention and incontinence in this region.

The book is full of original observations which will be of the greatest interest not only to urologists and general surgeons but to physiologists and pathologists ; we will try to pick out a few striking examples.

In cases of a stone lying free in the pelvis, an incomplete evacuation of the pelvis was almost always noted ; this is not due, as generally taught, to mechanical obstruction, but to spasm or atony of the pelvic muscle. Incomplete evacuation was also noted in some, though not in all, cases of movable kidney ; but this, though accompanied by a kink in the ureter, is not caused by the kink, for the arrest occurs not at the kink but always at the level of the pelvis ; a similar condition obtains in cases of obstruction by an abnormal renal vessel, for, though the evacuation is incomplete, the arrest again occurs at the pelvis and not at the level of the vessel, which is beautifully shown as a transverse filling defect.

In all these three examples, the authors consider that the retention is not mechanical but is due to the presence of what they call an 'épine irritative', a picturesque wording for an exciting cause ; this they think suffices to throw the pelvic muscle into spasm or to cause atony if sufficiently prolonged.

The book is well and clearly written, the pyelograms are excellently reproduced, and the legends describing the latter are also given in English, Spanish, Italian, and German. We offer our hearty congratulations to the authors, and strongly recommend our readers who are interested in the subject of kidney surgery to study this stimulating monograph.

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EPOCH-MAKING BOOKS IN BRITISH SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

VI. JOHNSON'S AMBROSE PAREY.

JOHNSON'S translation of "The Workes of that famous Chirurgion Ambrose Parey out of Latin and compared diligently with the French" made the book a working guide for English surgeons from the appearance of the first edition in 1634 until at least the end of the century. The surgeons of this country took it at once to their hearts and quoted freely from it in season and out of season, for Johnson had made it speak in such homely English that it is difficult when reading it to think that it is merely a translation. The need of English surgeons for a trustworthy and detailed text-book on surgery was very great at the beginning of the reign of King Charles I. The works of the Tudor surgeons, as has been shown, merely gave details of their own somewhat limited experience. Woodall wrote especially for the guidance of medical officers serving at sea or on land: Johnson translated for the benefit of civil surgeons as well as for those attached to armies. The book was a perfect mine of information on all subjects, even on those only remotely connected with surgery. "Save Art and Politics," says Mr. Stephen Paget, "the works of Paré contain every possible subject; Anatomy and Physiology, Medicine, Surgery, Obstetrics, State Medicine, Pathology, Pharmacy, Natural History, Demonology and much else. The divine origin of diseases, the influence of the stars, the power of devils, the nature of the soul, the history of medicine—he ranges from these to the tricks of beggars and of quacks, the homely remedies of old women, the folly of tight-lacing, the best sort of tooth powder and the right way to make pap for a baby." Best of all perhaps is the trouncing of Etienne Gourmalen, Dean of the Faculty of Medicine at Paris. Here was really good reading for a winter's evening when as yet there were no newspapers, no novels, and the romances were too long and too dull to interest a tired surgeon.

Of Ambroise Paré the great French surgeon (1509–90) we know much; of Thomas Johnson his translator comparatively little, although he carried

into the seventeenth century the grand tradition of the translators of the Elizabethan period—translators who, whilst adhering fairly closely to their texts, were able to render their authors' writings into the nervous English which it is still a joy to read. Johnson appears first as an apothecary with a physie garden in Snow Hill close to St. Bartholomew's Hospital; then as a botanist of repute, and with such a knowledge of plants that he was able to edit, correct, and largely augment Gerard's Herball; lastly as a Royalist Lieutenant-Colonel of Horse who was wounded in the defence of Basing-House, where he received a shot in the shoulder on Sept. 14, 1644, "whereby contracting a fever he died a fortnight after, his worth challenging funerall tears being no less eminent in the garrison for his valour and conduct as a soldier than famous through the kingdom for his excellence as an herbarist and physieian." It is recorded of him that "when a dangerous piece of service was to be done, this doctor, who publicly pretended not to valour, undertook and performed it." In 1633 he published Gerard's Herball, a folio of 1630 pages with innumerable drawings of plants; in 1634—the very next year—the "Works of Ambrose Parey" appeared, a folio of 1173 pages also illustrated with hundreds of cuts. During those years his house must have been full of proof sheets and positively littered with pulls of the blocks, each of which had to be allotted to its proper place. How was it done and what hours did Johnson keep? Philemon Holland's work was mere child's play in comparison, for his Pliny, Livy, Plutarch, and Schola Salernitana have no illustrations, so everything was plain sailing. There is, however, some evidence to show that Johnson did not work single-handed. Parey was on the stocks for some time, because a licence was taken out for the book at Stationers' Hall as early as September, 1629. The "Book of Apologies and Voyages", too, which appeared after the Latin translation attributed to Guillimeau, from which Johnson worked, was not issued until 1585. It was therefore especially translated out of the French by George Baker, who was, Johnson says, "a surgeon of this City since that time, as I heare, dead beyond the seas."

George Baker was Serjeant Surgeon to Queen Elizabeth, and at one time Master of the United Company of Barbers and Surgeons of London. He did some good literary work in his day and was a friend of Gerard, who had served as one of the public examiners during his Mastership. He seems to have been a man of violent temper, for there is a minute in the records of the Barber-Surgeons' Company dated "25 March 1577. Here at this Court was a great contention and strife spoken of and ended between George Baker and William Clowes for that they both contrary to order and the good and wholesome rules of this house misused each other and fought in the fields together. But the Master, Wardens and Assistances wishing that they might be and continue loving brothers pardoned this their great offence in hope of amendment." Johnson's note has by some accident been overlooked by those who have recorded the facts of George Baker's life. Nothing was known about him after he left the Company except that he died in 1609. It is clear that he had been living in France and that he had a competent knowledge of the language.

Johnson published a second edition of Gerard's Herball in 1636, but the



THE
 WORKES
 of that famous
 Chirurgion
 Ambrose Parey
 Translated out of
 Latine and compared
 with the French.
 by Tho: Johnson.

Whereunto are added three Tractates
 out of Adrianus Spigelius of the
 Veines, Arteries, & Nerves,
 with large Figures.

Also a Table of the Bones and Chapitres.

London .:s
 Printed by Richard Cotes, and
 Willi Dugard, and are to be sold by Joh:
 Clarke, entering into Mercers Chappell, 1673.



second edition of Paré's Surgery did not appear until 1649, and by that time the gallant and industrious translator had been dead for five years.

The following extracts show the value of the information given and the vigour of the translation. Paré's surgical training was almost entirely gained in camps, but he was able to adjust his teaching to the requirements of surgeons in civil practice. Thus, speaking of the reduction of a dislocated shoulder he says: "The patient must be layd with his backe on the ground upon a Cover-lid or Mat, and a clew of yarne or leathern-ball stuffed with tow or cotton, of such bignesse as may serve to fill up the cavities must be put under his arm-pit, that so the bone may straight-wayes the more easily be forced by the heele into its cavity. Then let the Surgeon sit beside him, even over against the luxated shoulder; and if his right shoulder be luxated, he shall put his right heele to the ball which filled up the armpit; but if the left, then the left heele. Then let him forthwith draw towards him the Patients arme, taking hold thereof with both his hands and at the same instant of time strongly presse the arme-pit with his heele. Whilst this is in doing one shall stand at the Patients backe, who shall lift up his shoulder with a towell, or some such thing fitted for that purpose, and also with his heele presse downe the top of the shoulder-blade; another also shall sit on the other side of the Patient, who, holding him, shall hinder him from stirring this way or that way at the necessary extension in setting it." He further teaches that the head of the humerus may be displaced into the armpit, outwards or forwards.

Paré draws attention to the metastatic abscesses which occur in pyæmia, a complication which does not seem to have been noticed previously. He says, speaking by the mouth of Johnson: "In these late civill warres the wounds which were for their quantity small, for the condition of the wounded parts but little, have caused so many and grievous accidents and lastly death itself. Now there came such a stinke, which is a most assured signe of putrefaction, from these wounds when they were dressed that such as stood by could scarce endure it, neither could this stinke bee attributed to the want of dressing or fault of the Chirurgion, for the wounds of the Princes and Nobility stunk as ill as those of the common Souldiers. And the corruption was such that if any chanced to bee undrest for one day, which sometimes happened amongst such a multitude of wounded persons, the next day the wound would be full of wormes. Besides also, which furthermore argues a great putrifaction of humors, many had Abscesses in parts opposite to their wounds, as in the left knee when as the right shoulder was wounded; in the left arme when as the right Leg was hurt. Which I remember befell the King of Navarre, the Duke of Nevers, the Lord Rendan and divers others. For all men had nature so overcharged with abundance of viciois humors that if it expelled not part thereof by impostumes [abscesses] to the habite of the body it certainly otherwise disposed of it amongst the inner parts of the body, for in dissecting dead bodies wee observed that the Spleene, Liver, Lungs and other Bowells were purulent and hence it was that the patients by reason of vapours sent from them to the heart were troubled with continuall feavers. But the Liver and all the veinous parts being polluted and so the generation of the laudable blood hindered, they languished for want of

fitting nourishment. But when the Braine by vapours was drawne in to sympathize with the rest they were molested with Ravings and Convulsions. Wherefore if any thing succeeded unprosperously in so great malignancie of wounds the Chirurgeon was not to be blamed for that it were a crime to fight against God and the Aire wherein the hidden scourges of the divine justice lye hid." Truly a comfortable doctrine from the surgeon's point of view.

Paré recognized clearly fracture of the skull by contrecoup, and he devotes a chapter to "Resonitus or Counter-fissure", in his book "of the Greeue and Bloody Wounds of each Part" saying: "Sometimes the fracture is made in the part opposite to that which received the blow; as if the right side be strueke the left is cloven. This kinde of fracture is very dangerous because we cannot finde it out by any certaine signe. Wherefore, if at any time the patient dye of such a fracture, the Chirurgion must be pardoned. This shall be made manifest by recitall of the following historie,—A servant of Massus the postmaster, had a greevous blow with a stone upon the right Bregma, which made but a small wound yet a great contusion and Tumour. Wherefore, that it might more plainly appeare whether the bone had received any harme and also that the congealed blood might be pressed forth, the wound was dilated, the skinne being opened by Theodore Hereus, the Chirurgion, who as hee was a skillful workeman and an honest man omitted nothing which Art might doe for his cure. When he had divided the skinne the bone was found whole, although it was much to bee feared it was broken because he fell presently to the ground with the blow, vomited and showed other signes of a fractured scull; so it happened that he dyed on the one and twentieth day of his sicknes. But I, being called to learne and search how he came by his death, deviding the scull with a saw, found in the part opposite to the blow a great quantity of sanies or bloody matter and an Abscesse in the Crassa meninx [dura mater] and also in the substance of the very braine but no sutures but the two scaly ones. Therefore, that is certaine which is now confirmed by the authority of Hippocrates as also by reason and experience, that a blow may bee received on the one side and the bone may be fractured on the opposite, especially in such as have either no sutures or else so firmly united and closed that they are scarce apparent."

The next extract is hackneyed, but it still bears repetition. It tells of the way in which Paré was driven to use a simple dressing for gunshot wounds: "In the yeare of our Lord 1536 [he was born in 1509 or 1510] I was in the Kings Army, the Chirurgion of Monsieur Montejan, Generall of the foote. I will tell the truth. I was not very expert at that time in matters of Chirurgery, neither was I used to dresse wounds made by Gunshot. Now I had read in John de Vigo that wounds made by Gunshot were venenate or poisoned and that by reason of the Gunpowder. Wherefore, for their cure it was expedient to burne or canterize them with oyle of Elders, scalding hot, with a little Treacle mixed therewith. It chanced on a time that by reason of the multitude that were hurt. I wanted this Oyle. Now because there were some few left to be dressed I was forced, that I might seem to want nothing and that I might not leave them midrest, to apply a digestive made of the yolke of an egge, oyle of Roses and Turpentine. I could not sleepe all that night for I was troubled in minde and the dressing of the precedent day

(which I judged unfit) troubled my thoughts; and I feared that the next day I should finde them dead or at the point of death by the poyson of the wound, whom I had not dressed with the scalding oyle. Therefore I rose early in the morning, I visited my patients and beyound expectation I found such as I had dressed with a digestive onely, free from vehemencie of paine, to have had good rest and that their wounds were not inflamed nor tumified but, on the contrary, the others that were burnt with the scalding oyle were feaverish, tormented with much paine and the parts about their wounds were swolne. When I had many times tryed this in divers others I thought this much, that neither I, nor any other, should ever cauterize any wounded with Gunshot." Thus began a revolution in the treatment of gunshot wounds, not all at once though, for the method was not generally adopted, and Paré himself preferred a balsam of boiled whelps and earthworms to the simpler dressing which necessity had compelled him to use with such good results.

To stop bleeding after amputation he recommends that: "The ends of the vessells lying hid in the flesh must be taken hold of & drawn, with this instrument [a pair of Crow's Beak forceps], forth of the muscles where into they presently after the amputation withdrew themselves. as all parts are still used to withdraw themselves, towards their originalls. In performance of this worke you neede take no great care if you, together with the vessells, comprehend some portion of the neighbouring parts, as of the flesh, for hereof will ensue no harme, but the vessells will bee consolidated with the more ease than if they, being bloodlesse parts, should grow together by themselves. To conclude, when you have so drawne them forth binde them with a strong double thred."

"Verily," he goes on to say in another chapter, "I confesse I formerly have used to stanch the bleeding of members after amputation after another manner than that I have a little before mentioned. Whereof I am ashamed and agreived. But what should I doe? I had observed my maisters, whose methods I intended to follow, alwaies to doe the like; who thought themselves singularly well appointed to stanch a flux of blood when they were furnished with various store of hot Irons and causticke medicines which they would use to the dismembred part, now one, then another, as they themselves thought meete. Which thing cannot be spoken or but thought upon without great horror much lesse acted. For this kinde of remedy could not but bringe great and tormenting paine to the patient, seeing such fresh wounds made in the quicke and sound flesh are endowed with exquisite sense. Neither can any causticke be applyed to nervous bodies but that this horrid impression of the fire will be presently communicated to the inward parts whence horrid symptomes ensue and oftentimes death it selfe. And verily of such as were burnt the third part scarce ever recovered and that with much adoe for that combust wounds difficultly come to cicatrisation; for by this burning are caused cruell paines whence a Feaver, Convulsion and oft times other accidents worse than these. Adde, hereunto, that when the eschar fell away oft times a new hæmorrhagye ensued for stanching whereof they were forced to use other causticke and burning Instruments. Neither did these good men know other course so by this repetition there was great losse and waste made of the fleshy and nervous substance of the part. Wherefore I

most earnestly entreate all Chirurgions that leaving this old and too too cruell way of healing they would embrace this new, which, I thinke, was taught mee by the speciall favour of the sacred Deitie, for I learnt it not of my maisters, nor of any other, neither have I at any time found it used by any." These brave words and good advice were wasted in England, for in London the cautery was certainly in use to stop bleeding after amputation until 1745.

The title page, of which a slightly reduced copy is here given, is identical in the editions of 1634 and 1649. It was engraved by Thomas Cecil, of whom very little is known except that he lived and worked in London from 1627 until 1635.

AN OPERATION FOR MAKING THE FOREARM PREHENSILE AFTER LOSS OF A HAND.*

By ARNOLD K. HENRY,

PROFESSOR OF CLINICAL SURGERY IN THE FACULTY OF MEDICINE, CAIRO.

CERTAIN of the more distal amputations have the effect of putting the muscular engine of a limb permanently out of gear; the contractile bellies remain ineffectual but intact, and from time to time surgical endeavour has been stimulated to exploit this waste of power. A large and accessible literature deals with the group of operations based upon the work done by

Vanghetti on birds, which revealed the fact that tendons properly clothed with skin can be made, for example, to pull strings and work the fingers of artificial hands. Apart, however, from this cinemaplastic group, the record of muscular exploitation in amputated limbs is small, and not everywhere available. My only guides to the bibliography have been two papers in Russian by Rosanoff and Geimanovitch which I owe to the courtesy of my colleague, Dr. Boris Boulgakow, Curator of the Museums of the Faculty of Medicine, Cairo, who received them some time after I had performed the operation which is the subject of this study. Both Russian surgeons give accounts of cases treated by a method published by Krukenberg, of Stuttgart, in 1917.

I feel it not merely a duty but a privilege to make Krukenberg's operation more widely accessible; and to the following description of my own procedure, which was completed in ignorance of Krukenberg's work,

I have added an account of his method which I have taken from Dr. Boulgakow's translation.

My interest in the problem of putting the muscles of an amputated



FIG. 177.—Radiogram of the stump of the left forearm before operation.

* From the Surgical Unit, Kasr el Aini Hospital, Cairo.

stump back into gear was aroused by the admission to my Surgical Unit on April 12, 1927, of an Egyptian policeman whose left hand had been cut off at the wrist by a tramcar wheel (*Fig. 177*). His age was 22. When I first saw him the stump was painful and septic. The sepsis, however, was soon controlled; healing began, and it seemed a pity that the muscular energy of his well-developed forearm should be wasted for lack of surgical effort. I therefore outlined the following procedure which I practised on the cadaver, and though it did not eventuate precisely 'according to plan', I feel that something may be learnt by a comparison of what was intended with what actually was accomplished.

My objects were the following: (1) To cut from the outer side of the radius a rod of bone which should have about the total length of a normal thumb, including the metacarpal segment; (2) To leave *in situ* (*a*) the thumb tendons already related to this piece of bone, i.e., the long abductor (O.T. ext. ossis metacarpi pollicis) and short extensor (O.T. ext. primi internodii pollicis), and (*b*) the tendon of the brachioradialis; (*c*) the trunk of the radial artery; (3) To make two false joints, one in the rod, about two inches from the distal end, the other proximally where the rod should meet the radius; (4) To attach two flexor tendons to the jointed rod, viz., the flexor pollicis longus and the flexor carpi radialis; (5) To enclose these structures in a tube of skin; (6) To set the new digit thus framed and furnished obliquely across the palmar face of the stump in such a way as to oppose its ulnar border. I hoped in this manner to provide the maimed limb with a sentient and prehensile mechanism, in appearance like the light glove used in a prize-fight.

The initial difficulty before me was to secure a clean field for osteoplastic operation. I was certain from experience that once the patient was allowed to leave the hospital with a cicatrized stump, resignation would set in and he would never return. I did not, therefore, wait for the stump to heal, but decided to attempt the destruction of latent sepsis by means of the high-frequency current, while the patient was yet willing for operation.

THE OPERATION.

On June 22, 1927, I completed at one sitting what was in effect a two-stage intervention. The limb received the usual picric acid preparation on two successive days.

STAGE I.—I first charred the granulations of the stump, together with an inch-wide margin of skin, with sparks from a high-frequency current. I then coagulated the region of the carpus. The limb was cleaned afresh with ether and 5 per cent solution of picric acid in alcohol, and after change of gloves and instruments the second stage of the operation was begun.

STAGE II.—A circular disarticulation was made at the radiocarpal joint, dividing the skin a finger-breadth distal to the radial styloid. Two longitudinal 5-in. incisions were then made in the forearm—one in the mid-line of its flexor aspect, the second on the dorsal side a finger-breadth radial to the middle line. In order to provide a 'web' at the base of the new digit, both these incisions approached the ulna at their proximal ends.

Each cut was then deepened to the bone—the volar between the tendons of

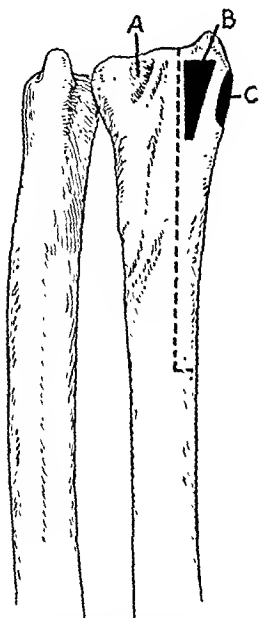


FIG. 178.—Diagram showing the part of the radius used to form the skeleton of the new digit. The broken line indicates the saw cut which separates the bony rod. The two black patches mark the two grooves which lodge (1) the radial wrist extensors, and (2) the two thumb tendons. After operation these grooves became points of insertion. (The normal insertion of the brachioradialis is close to the smaller patch.) A, Groove for extensor pollicis longus; B, Groove for radial wrist extensors; C, Groove for abductor longus and extensor pollicis brevis.

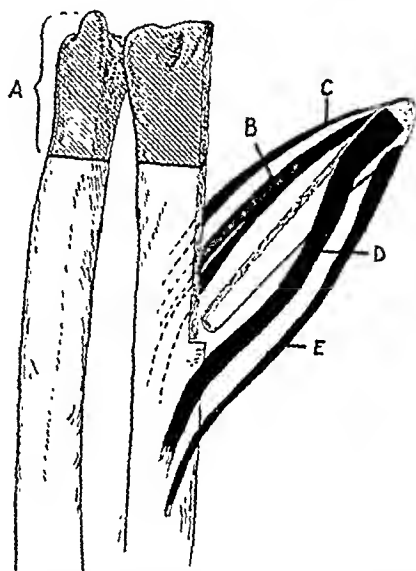


FIG. 179.—Diagram showing the separation of the bony rod from the radius. The rod carries with it the radial wrist extensors, two dorsal thumb tendons, and the brachioradialis. Two flexor tendons and the radial artery are added *en bloc* to the rod. (For simplicity, the five dorsal tendons are grouped into two single bands in the figure.) The radius and ulna were shortened by resecting the parts cross-hatched in the diagram: this allowed a medial flap of skin to be turned over the cut ends. (See Figs. 180, 181.) A, Parts of radius and ulna to be resected; B, Flexor pollicis longus; C, Flexor carpi radialis; D, Radial wrist extensors; E, Dorsal tendons of thumb with brachioradialis.

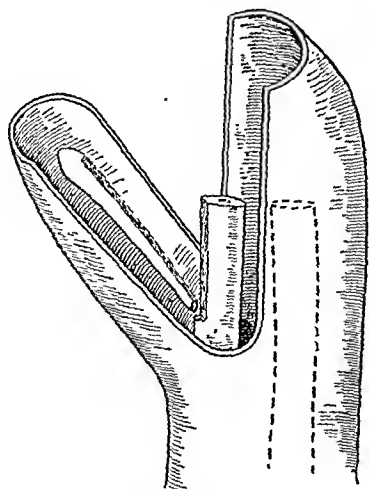


FIG. 180.—Diagram showing the skin flaps. The smaller flap is sutured round the bony rod; the larger is turned down over the ends of the ulna and radius. The result is like a light boxing glove (see Fig. 181).

the flexor carpi radialis and the flexors of the fingers, the dorsal between the radial extensors of the wrist and the common extensors of the fingers. Special care was taken to preserve intact the bellies of the abductor pollicis longus (O.T., ext. ossis metacarpi pollicis) and the extensor pollicis brevis (O.T., ext. primi internodii pollicis) which crossed the line of incision and were retracted proximally. The periosteum was divided longitudinally, and at the distal end of the radial shaft a 5-in. rod was separated from the lateral third of the bone by means of a motor-driven saw (Fig. 178). An attempt was thus

made to obtain a joint in the bony rod 2 in. from its distal end by dividing it subperiosteally, with cuts so placed as to prevent over-extension.

While these cuts were being made the proximal segment of the bony rod became detached from its periosteum and fell out upon the sterile towels round the wound; it was immediately replaced and the periosteum was stitched round it with catgut. Fortunately this accident gave rise to no subsequent trouble.

The tendon of the flexor pollicis longus, the radial artery, and the tendon of the flexor carpi radialis were transferred *en bloc* to the flexor face of the rod (Fig. 179). The skin was then stitched round the new digit. The ulnar and radial shafts were next shortened sufficiently to allow a medial flap of skin to be turned like a hood over their radial aspect (Fig. 180), and when this flap was sutured into place the resemblance of the end of the limb to the prize-fighter's glove was striking.

It is perhaps worth noting here that the rough likeness to a fist so pleased the owner that he was at first content to wear his limb as an ornament without attempting to use it (Fig. 181).



FIG. 181.—The right and left upper limbs two months after operation, showing the new digit. The clear band in the tattooing is over the radio-carpal joint of the normal right limb. The left ulnar shaft has been shortened by 5.5 cm. The length of the new digit measured along its medial side is 7 cm.; its circumference is 9.5 cm. The maximal angle between the digit and the stump is 65°; this angle can be reduced, by pure pronation, to 27°, giving an effective range of movement through 38°.

POST-OPERATIVE COURSE.

This was complicated by two minor mishaps: (1) The edge of the medial flap, which was turned over the radius and ulna, must, I think, have been slightly cooked in the thorough coagulation of the carpal region, and there was some sloughing at its surface. Fortunately this did not lead to any deep infection, which in this place would have jeopardized the skeleton of the new digit. (2) The cut end of the radial shaft should undoubtedly have been rounded off more carefully, for three weeks after the operation an area of pressure necrosis the size of a sixpenny piece appeared in the skin over it, and on Sept. 7, 1927, a small sequestrum was removed.

Just at this time, too, a sudden shortage of staff made it difficult to give the patient the full after-treatment he would otherwise have received, and I am most grateful to the succession of sisters on temporary duty who under

an exceptional pressure of routine work did so much for the education of the reconstructed limb.

In about a month free passive movement could be made at the distal joint of the new digit, while slight active movements were seen at the proximal joint, and many hours were spent in trying to develop them. The patient took little interest in these attempts, and for some three months, apart from the brief daily periods which could be spared for his tuition, he made no serious attempt to use the limb. One day, however, after faradic stimulation, he surprised me by making a few large movements of the new digit.



FIG. 182.—Radiogram of new digit 81 days after operation.

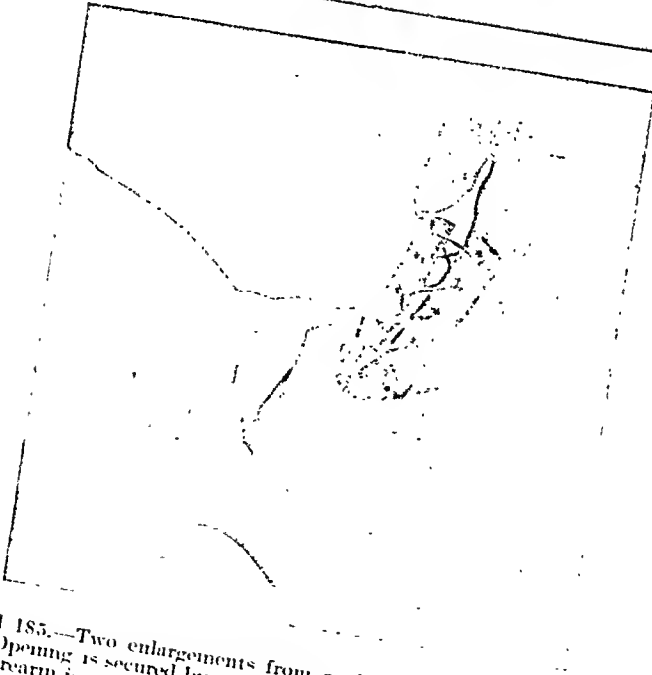
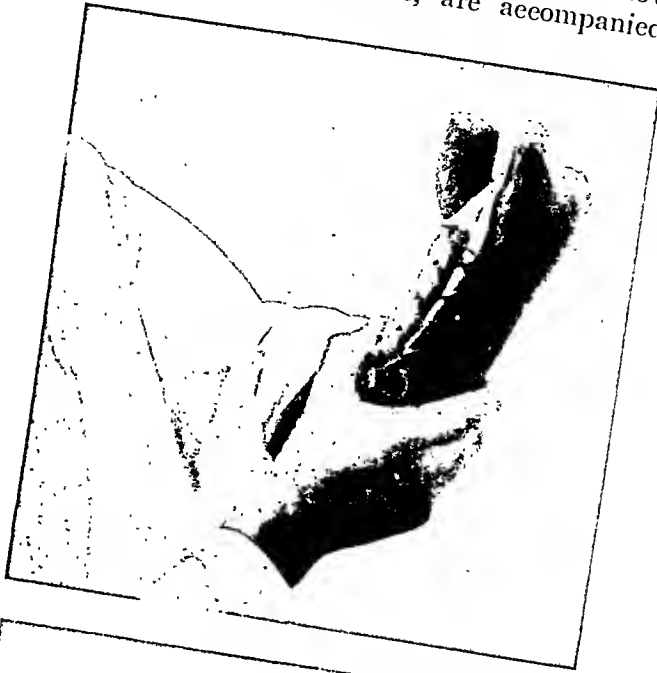


FIG. 183.—Radiogram of new digit 133 days after operation.

At first he could only reproduce these movements occasionally—at one moment they would be present, at the next absent; but soon after, he began to make them at will, and for a while I was under the mistaken impression that they were taking place at the intended joint between the radius and the proximal end of the bony rod. It will be seen, however, from the radiograms (*Figs. 182, 183*) that the rod had become ankylosed with the radial shaft. The deception lay in the unexpected mobility of the radius in the stump, and careful examination was needed to convince oneself that the new digit did *not* move independently. Actually, it was carried round into opposition by the pronation of the radius, while supination restored it to the position of rest (*Figs. 184, 185*). During these two active phases the tendons inserted into the digit stood out sharply.

PREHENSILE FOREARM AFTER LOSS OF HAND 193

It is relevant to note that in the normal hand a movement of foreible and sudden opposition, and its reverse, are accompanied respectively by



FIGS. 184 and 185.—Two enlargements from a cinematograph film showing the 'grip' open and shut. Opening is secured by supination; shutting, by pronation. The prothesis laced round the forearm is provided with a rigid artificial 'little finger' against which the new digit can press.

slight degrees of pronation and supination. The patient, therefore, in utilizing his new digit, merely promoted an associated movement to the rank of the prime movement itself, and, once his brain had acquiesced, a real progress

began. This was accelerated by Miss Gwillim, masseuse to the hospital, who then returned from leave and was able very soon to teach the patient to grasp a small matchbox between the new digit and the palmar face of the stump.

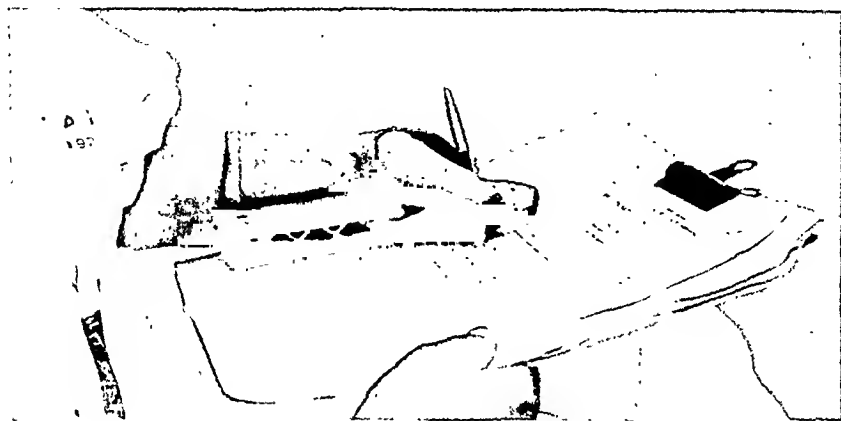


FIG. 186.—The patient writing with his left forearm.



FIG. 187.—Enlargement from a cinematograph film showing the patient after biting a piece of bread held with the left forearm.

This grasp was weak and could only be accomplished at the root of the digit, the real strength of which was wasted because—to revert to my former

illustration—the palm of the boxing glove was too short to allow of contact with the thumb. It became obvious that some form of apparatus was needed to give the digit a kind of little finger against which it could be forcibly opposed, and after consultation with my colleague, Professor A. Biggam, I designed a gauntlet to fit over the stump and lace round the forearm, leaving the new digit free (*Fig. 184*). This design was carried out for me by Mr. Moring, of Cairo, to whom I am indebted for his careful co-operation. Once the gauntlet was fitted the whole mentality of the patient seemed suddenly to change; his apathy vanished, and at once he began to work hard and continuously to master his new grip. Within forty-eight hours he could write a fair Arabic script with his prehensile stump, and a week later, just three months after operation, he employed the limb to perform a variety of functions, one of which is clearly seen in *Fig. 187*.

COMMENT.

In orthopædic surgery a simple procedure is often the best, and I feel on reflection that my attempt to construct a two-jointed thumb-like organ erred on the side of complexity. The clear lesson I have learnt from the intervention is the fact—which I had noted beforehand as a possible event—that the movement of pronation may be made to impart grip to a stiff digit fixed at an angle to the radius. Should occasion offer, I would incline, in the present state of my knowledge, to utilize this coarse but powerful movement rather than try to reproduce the fine machinery of the hand. With this aim now clear I should remove all the radial shaft distal to the attachment of the digit, and by procuring a partial fracture of the ulna I should bend it—like a little finger—in such a way as to give the radial digit a living opponent. This would dispense with the prothesis.

Krukenberg's Operation.—The following description of Krukenberg's operation, which I owe to Dr. Boulgakow's translation, throws light, from another angle, on a difficult problem.

Krukenberg's object is to convert the radius and ulna into the two jaws of a 'crocodile' forceps; Rosanoff gives the following description of his method.

Incision.—The line of incision is kept close to the ulna and begins on the volar surface of the forearm at a point 7 cm. distal to the bend of the elbow. From thence it passes longitudinally and turns round the end of the stump to a point at the same level on the dorsal surface. This gives a U-shaped cut which is deepened so as to separate the flexor digitorum sublimis into a radial and ulnar moiety (*Fig. 188*).

Resection of Nerves and Muscles.—The median and ulnar nerves are cut short, taking care not to damage their branches to useful muscles. The flexor pollicis longus and the flexor digitorum profundus are entirely resected in order to make the two jaws of the forceps less bulky and more easy to clothe with skin.

Division of the Interosseous Membrane.—This membrane is divided for a sufficient distance to obtain a separation of 12 cm. between the ends of the

radius and ulna. In making this cut the knife should be kept close to the ulna, and the interosseous arteries should if possible be spared.

Treatment of the Bones and Remaining Muscles.—The radial moiety of the flexor sublimis is sutured to the flexor carpi radialis, and the ulnar moiety to the ulnar flexors. The extensors are similarly united into radial and ulnar groups. The extremities of the radius and ulna are freshened by removing a centimetre of bone from each shaft, and their ends are grooved at right angles to the volar surface of the limb. The radial flexors are joined with the radial extensors in such a way that the junction between them lies in the groove at the end of the radius; the ulnar flexors and extensors are similarly dealt with.

Skin Closure.—Owing to the ulnar position of the skin incision, closure is easily effected round the radial jaw of the forceps.* A raw area remains on the ulnar part, and this is grafted, either at once or later, by means of a pedicled flap cut from the abdomen.

Movements are begun as soon as healing is complete, and are supplemented by the usual routine of massage and electrical treatment. One of Rosanoff's two patients—a student who knew anatomy—was able to write with a thick pen a month after operation.

Geimanovitch states that Bosch Arana and Schelle† have worked out the precise way in which the muscles function in the Krukenberg limb. Rosanoff finds that the abduction of the radial limb of the forceps is due to the brachioradialis muscle, the flexor carpi radialis, and one half of the flexor digitorum sublimis, and that adduction is

procured by the pronator teres, flexor carpi radialis, and the other half of the flexor digitorum sublimis.

The points of difference between Krukenberg's procedure and my own may be summarized as follows: (1) Krukenberg converts the entire remnant of the radius into the moving jaw of a 'crocodile' forceps. Division of the interosseous membrane allows the radius to be adducted and abducted at the radio-humeral joint. My grip was obtained by setting part of the radius



FIG. 188.—Krukenberg's method. The anatomy of the 'crocodile' forceps, showing the separation of the muscle groups after dividing the interosseous membrane. The flexor digitorum sublimis (J) is separated into two moieties; the flexor digitorum profundus and flexor pollicis longus have been resected. A, Biceps; B, Brachialis; C, Supinator; D, Brachioradialis; E, Pronator teres; F, Palmaris longus; G, Flexor carpi radialis; H, Flexor carpi ulnaris; J, Flexor digitorum sublimis. (After Rosanoff.)

* Rosanoff alludes to a modification of Krukenberg's technique which was proposed by Flockemann, who favours bayonet-shaped incisions. Rosanoff thinks that, provided the bones were shortened sufficiently, this would give a complete covering of skin to the ulnar segment. This agrees with my own experience.

† Or Scheel (?). Both spellings occur in Geimanovitch's paper.

at an angle to the rest and so transforming the movement of pronation into one of opposition. (2) Krukenberg obtains a supplementary covering of skin from the abdomen. In my case a complete skin covering was secured from the forearm itself.

Further experience alone can decide the respective indications for the use of these different principles. Possibly requirements of strength as opposed to delicacy of movement may influence the choice. In these matters much depends upon the patient himself, and in claiming merit for an orthopædic measure it is well to remember the Arabic saying, "The clever spinner spins with an ass's foot".

My thanks are due in a special degree to Dr. Ahmed Handousa, formerly Senior Surgical Registrar at Kasr el Aini Hospital, for the constant personal care which he devoted to this case and for his assistance in the preliminary work on the cadaver. I owe to him the suggestion, made during the operation, of shortening the bone to obtain an immediate covering of skin. I am indebted, too, to Dr. Boulgakow, who, besides interpreting the literature and providing the entire bibliography, has redrawn for me the figure of Krukenberg's operation which is taken from Rosanoff's paper. I owe the radiograms to my colleague, Dr. R. A. Gardner.

BIBLIOGRAPHY.*

- VANGHETTI, *Plastica e protesi cinematiche*, 1906.
 KRUKENBERG, *Ueber plastische Umwertung von Amputationsstümpfen*, Stuttgart, 1917.
 FLOCKEMANN, *Beitr. z. klin. Chir.*, 1923, xvii.
 ROSANOFF, *Nov. Archiv. Chir.*, 1923, i.
 FAERMAN, *Jour. Theoret. i Prakt. Med.*, 1925, i.
 GEIMANOVITCH, *Vratchenevje Delo*, 1927, April, No. 8.
 SCHEEL, *Zeits. f. d. orthop. Chir.*, 1920, xxxix.
 BOSCH ARANA, *Prensa méd. Argentina*, 1923, No. 1.

* These references, with the exception of Vanghetti's publication, are quoted from the papers of Rosanoff and Geimanovitch; the journals are not available in Cairo.

A CASE OF PRIMARY CARCINOMA OF THE BRONCHUS, WITH SECONDARY BRONCHIECTASIS : DEATH FOLLOWING PHRENIC EVULSION.*

BY MAURICE DAVIDSON,

PHYSICIAN, MILLER GENERAL HOSPITAL FOR SOUTH-EAST LONDON ;

AND REGINALD C. B. LEDLIE,

ASSISTANT SURGEON, MILLER GENERAL HOSPITAL, LONDON.

THE patient, a lean, spare-built man of 48, was admitted to the Miller Hospital on Jan. 1, 1928, on account of a recent hæmoptysis. He said that he had always enjoyed good health and had never been absent from work through illness. Early in December, 1927, he developed a slight cough, and shortly after Christmas he expectorated a very small quantity of blood mixed with sputum. Three days later he had a more definite hæmoptysis which, though not severe, was sufficient to alarm him, and he came to hospital for advice and was admitted for observation. After a few days the hæmorrhage had entirely ceased, but he continued to expectorate a fair amount of nummular sputum. No tubercle bacilli were found in this on two separate examinations. There was no dyspnœa or cyanosis, nor did he appear in any way distressed. The heart was normal in size and position. At the base of the right lung behind there was marked dullness, with diminished tactile fremitus and vocal resonance, and weak bronchial breathing; a few fine râles were audible at the end of inspiration. The physical signs on the left side of the chest were normal. On screening the chest the movements of the diaphragm were seen to be restricted on the right side, translucence at the base of the right lung being diminished. The skiagram (*Fig. 189*) showed a definite opacity at the right base. A subsequent skiagram taken after intratracheal injection of 25 c.c. of lipiodol (*Fig. 190*) gave a characteristic picture of a localized bronchiectasis in the right lower lobe, the appearances in the right upper zone and in the whole of the left lung being normal.

In view of the fact that the disease was thus definitely unilateral and localized, this was thought to be an ideal case for evulsion of the phrenic nerve, which operation was accordingly performed on Jan. 30 under local anæsthesia, $8\frac{1}{4}$ inches of the nerve being removed. The patient was in no way distressed at the time of the operation, which was a perfectly straightforward one, the phrenic nerve being found without difficulty in the normal anatomical situation. His condition remained quite satisfactory for the rest of the day, and throughout the following day until about 10 p.m., when the temperature, which had been normal after the operation, rose to 100° , and the pulse-rate increased to 120. At 4 a.m. on Feb. 1 the temperature rose suddenly to 104° (*Fig. 191*), and he complained of severe pain in the right side and became

* From the Miller General Hospital, London.

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cyanosed and very distressed. Strychnine injections were given four-hourly with atropine, and also heroin for the pain. The following day he appeared somewhat better, the cyanosis having disappeared and the acute distress having subsided. On examination of the chest the level of dullness had risen appreciably, and it was evident that the right side of the diaphragm was effectively paralysed. Beyond the alteration in the physical signs naturally to be expected after the operation there was nothing remarkable, and it was difficult to understand what had occurred to give rise to the sudden and quite



FIG. 189.—Skiagram showing opacity at the base of the right lung.

unusual symptoms. From this time onwards he became rapidly weaker, and his general condition gave rise to increasing anxiety, although no additional information was afforded by physical examination as to the nature of the complication which had arisen. He died on the night of Feb. 8, nine days after the operation.

POST-MORTEM EXAMINATION.—For the following account of the pathological findings we are indebted to our colleague, Dr. Weston Hurst:—

“At autopsy the right side of the diaphragm was definitely higher than the left. The left lung was in a condition of compensatory emphysema and projected over the mid-line of the chest; the heart was similarly displaced.

The right lung, loosely adherent to the chest wall above, was at the base firmly bound down by adhesions; the free surface of the visceral pleura was covered by a thick fibrinous exudate, accompanied by a few ounces of yellowish slightly turbid fluid. At the base the pleura was much thickened by dense fibrous tissue, which also radiated as bands through the lung substance. The



FIG. 190.—Skiagram after lipiodol injection, showing localized bronchiectasis in the right lower lobe.

lower divisions of the right bronchial tree were considerably dilated, particularly near the base. From the lowest point of the trachea near its bifurcation, down to the smaller bronchi of about the size of a quill, the mucous membrane was represented by a rough shaggy lining; this tissue extended alike into the bronchi of the upper, middle, and lower lobes, but was most obvious in the last situation. No masses of growth were present in either lung, but the lymphatic glands at the bifurcation of the trachea were enlarged and breaking down into a thick puriform fluid. Except for some congestion, the bronchi

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on the left side appeared normal; a thin mucopurulent secretion could, however, be expressed from some of the smaller branches. Cultures from the right lung yielded a pure growth of pneumococcus.

"The heart was covered with a fibrinous exudate. Early atheromatous lesions were present in the aorta. The spleen was enlarged and showed the appearance associated with septic infection. Both kidneys were in a condition of cloudy swelling, and in the cortex of the right organ a large wedge-shaped area of necrosis was seen.

"On microscopic section, the right bronchus and its branches were found to be lined by a squamous-celled carcinoma which had replaced the mucous membrane. There was early invasion of the lung tissue in the lower lobe, but for the most part the neoplasm was confined to the air-passages. Patches of bronchopneumonia were present at the base. Early invasion of some of the bronchial glands and advanced infiltration of those near the bifurcation of the trachea were noted. The wedge-shaped area in the kidney was one of acute necrosis due to bacterial embolism; masses of bacteria could be seen plugging all the vessels in this region."

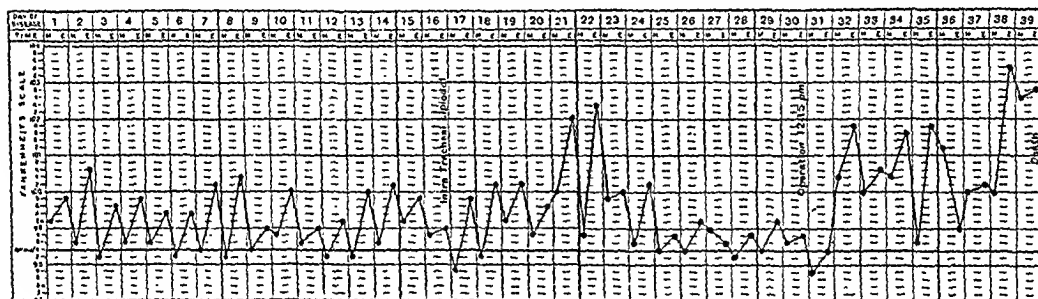


Fig. 191.

DISCUSSION.

It is not proposed to enter at length into the histological aspects of this case, the main interest and importance of which lie in its clinical features, but there are certain points which deserve notice. Tumours of the trachea and large bronchi are not of common occurrence, and according to MacCallum¹ are not ordinarily of the type of flat-celled epithelioma, since these canals are lined with cylindrical ciliated epithelium. In describing a series of squamous epitheliomata of the lung, MacCallum refers to the occurrence of 'prickle cells' undergoing keratinization, and thinks that the transition of cylindrical into squamous epithelium may be an example of metaplasia. A good instance of this has recently been published by Baden Evans.² The actual naked-eye appearances of the bronchus in our own case were interesting in that they were suggestive more of an inflammatory lesion than of new growth; and the microscopic section, which resembled that of any typical epithelioma involving the skin and showing characteristic cell nests, was something of a surprise.

For a general brief account of the main pathological and statistical data in regard to primary carcinoma of the lung and bronchi, reference may best

be made to Ewing.³ The statistical aspect of the subject is of considerable importance in view of the alleged increase in lung carcinoma in recent years. Some striking observations on this point are furnished by Hauf⁴ and by

Fried.⁵ Whether, as the latter seems to think, the increase is more apparent than real, is a point on which there is some difference of opinion. The present writers, without wishing to be dogmatic, incline strongly to the belief that the incidence of intrathoracic malignant growths has become considerably greater in this country in the last twenty years.

The clinical aspect of this case is of undoubted interest and practical importance. One of the salient points is the absence of any history of a previous pneumonia or of anything to suggest the presence of a foreign body in the bronchus; this was especially noted in the preliminary examination of the patient, in whom the occurrence of a localized bronchiectasis could not easily be accounted for. Had a bronchoscopic examination been made, the appearances illustrated in the coloured figure (*Fig. 192*) could easily have been seen, but it is doubtful if they would have established the diagnosis, unless one had been able to remove a portion sufficient to give a characteristic microscopic picture, which might have obviated the performance of what proved to be a needless operation. Even so the after-history is a little difficult to explain. Death was apparently due to the general dissemination of an intense pneumococcal infection in consequence of the sudden compression of the base of the lung following the paralysis of the diaphragm. In view of the fact that in about a hundred

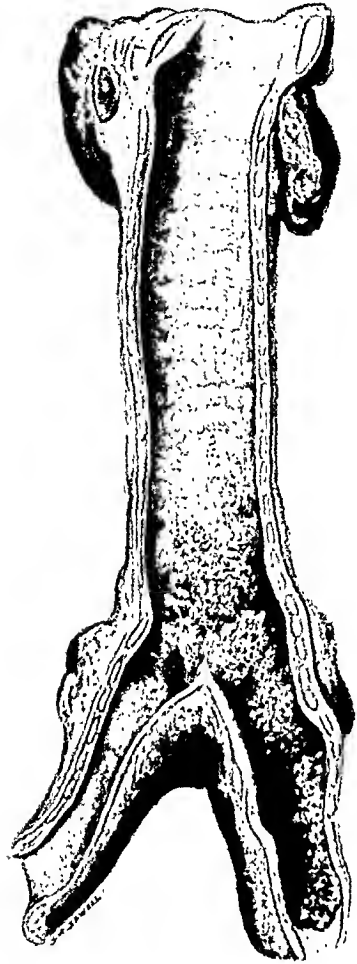


FIG. 192.—Carcinoma of bronchus.

instances of section or evulsion of the phrenic nerve of which one of us has had cognizance at the Brompton Hospital no untoward result has occurred, we have thought that the above case, apart from its interest from a diagnostic point of view, was of sufficient importance to merit a record in the literature of this operation.

REFERENCES.

- ¹ MACCALLUM, W. G., *Text-Book of Pathology*, 3rd ed., 1924, 1032.
- ² EVANS, D. M. B., *Lancet*, 1927, i, 1077.
- ³ EWING, J., *Neoplastic Diseases*, 3rd ed., 1928, 851.
- ⁴ HAUF, DORA, *Virchow's Arch.*, 1927, cclxiv, 366.
- ⁵ FRIED, B. M., *Arch. of Internal Med.*, 1927, xl, 340.

THE ACTION OF SALIVA AND GASTRIC JUICE ON THE CLOTTING OF BLOOD.*

BY JOHN B. HUNTER,

SURGEON TO THE ROYAL NORTHERN HOSPITAL, LONDON.

THE following small investigation was started in the belief that saliva had some action on shed blood, probably by decreasing the coagulation time. The rapidity with which wounds in the buccal cavity stop bleeding and the firm clots that occur in that situation seemed to indicate that saliva has a coagulant action. A further point was the common observation of the licking of their wounds by animals. On consideration the reasons for this fact appear briefly to be:—

1. *Cleansing of the Wound.*—The cleaning of the wound certainly does occur by means of the wet tongue's action, but whether this is a primary reason seems doubtful, as animals will frequently soil the wound again immediately after licking it.

2. *Covering the Wound with an Air-proof Dressing.*—This is done by means of the rather sticky saliva which coats the raw surface.

3. *To Relieve Irritation.*—In this case the action takes place at a later date, and no doubt the scratching action of the tongue alleviates irritation common to all healing wounds.

4. *To Bring Saliva into Contact with a Bleeding Surface.*—It is this last reason that is the subject of the following observations. As the second problematical reason for licking wounds I have given the production of an air-proof dressing to the wound by means of the viscid saliva, but it seemed probable that saliva might have a direct action on the coagulation of the blood, and that apart from its viscid character it might hasten coagulation. I attempted at first to get a pure secretion of saliva by means of a cannula tied into the salivary duct of a cat, and I carried out two experiments by this method, but found that considerable error was likely to occur in collecting the blood. I therefore took a simpler method. I collected as much of my own saliva as I required in a clean beaker, and drew off 20 c.c. of blood from the median basilic vein. The technique in all cases was the same. Paraffined test-tubes with paraffined corks were used, and the blood was drawn off into a paraffined syringe, the moment of clotting being estimated by the eversion of the tube. The results were as follows:—

CAT 1.				TIME TAKEN TO CLOT in minutes	
<i>Tube A.</i> —Saliva 1 c.c.					
Blood 3 c.c.	4 $\frac{3}{4}$
<i>Tube B.</i> —					
Blood 3 c.c.	7 $\frac{3}{4}$

* From the Surgical Unit Laboratory, University College Hospital.

CAT 2.

TIME TAKEN TO CLOT
in minutes*Tube A.*—Saliva $\frac{1}{2}$ c.c.

Blood 4 c.c. 3

Tube B.—

Blood 4 c.c. 10

In all the following experiments the saliva used was human, collected into a beaker.

EXPERIMENT 1.

TIME TAKEN TO CLOT
in minutes*Tube A.*—Saliva $\frac{1}{2}$ c.c. passed through
Berkefeld filterBlood 3 c.c. $13\frac{1}{2}$ *Tube B.*—Centrifuged saliva $\frac{1}{2}$ c.c.

Blood 3 c.c. 7

Tube C.—Saliva $\frac{1}{2}$ c.c.

Blood 3 c.c. 18

Tube D.—

Blood 3 c.c. 24

The total bulk in these experiments varied, and in the following experiments normal saline was added to the simple blood tube to make up the same bulk as in the other tubes.

EXPERIMENT 2.

TIME TAKEN TO CLOT
in minutes*Tube A.*—Centrifuged saliva 4 c.c.

Blood 4 c.c. 2

Tube B.—Saline 4 c.c.

Blood 4 c.c. 15

EXPERIMENT 3.

Tube A.—Centrifuged saliva 5 c.c.Blood 4 c.c. $3\frac{1}{2}$ *Tube B.*—Saliva 5 c.c.

Blood 4 c.c. 5

Tube C.—Saline 5 c.c.Blood 4 c.c. $16\frac{1}{2}$ *Tube D.*—Centrifuged diluted saliva,
1-4, 5 c.c.Blood 4 c.c. $3\frac{1}{2}$ EXPERIMENT 4 (the blood was not drawn
off into a paraffined syringe).*Tube A.*—Centrifuged saliva 3 c.c.Blood 3 c.c. $4\frac{1}{2}$ *Tube B.*—Saline 3 c.c.Blood 3 c.c. $8\frac{1}{2}$

These figures seem to show that the coagulation time of blood is very definitely reduced by the addition of saliva. Further, that rather unexpectedly the centrifuged saliva appears to act better than plain saliva, and that the passage through a Berkefeld filter does not alter the action.

The next point of interest in the investigation is the action of the gastric juice on the clotting of blood. This involved the double investigation of the action of gastric juice on the clotting time of blood alone, and in the presence of saliva. The nature of the experiments was the same as previously, the gastric juice being obtained from the results of fractional test-meals taken in the wards.

Preliminary experiments with *Cat 2* were done in which saliva, blood, and hydrochloric acid in two different strengths were mixed. The results of these seemed to show that clotting was markedly delayed with the stronger hydrochloric acid which was used (0.5 per cent).

The other experiments are as follows :—

EXPERIMENT 5.

TIME TAKEN TO CLOT
in minutes

<i>Tube A.</i> —Centrifuged saliva 4 c.c.	
Blood 4 c.c.	2
<i>Tube B.</i> —Centrifuged saliva 2 c.c.	
Gastric juice, 0.13% HCl, 2 c.c.	
Blood 4 c.c.	7
<i>Tube C.</i> —Centrifuged saliva, 2 c.c.	
Gastric juice, no HCl, 2 c.c.	
Blood 4 c.c.	7
<i>Tube D.</i> —	
Gastric juice, 0.13% HCl, 4 c.c. . .	{ Not clotted in 30 minutes Firm clot 55 minutes
Blood 4 c.c.	
<i>Tube E.</i> —Saline 4 c.c.	
Blood 4 c.c.	15

EXPERIMENT 6.

<i>Tube A.</i> —Centrifuged saliva 5 c.c.	
Blood 4 c.c.	3½
<i>Tube B.</i> —Saliva 3 c.c.	
Gastric juice, 0.2% HCl, 2 c.c.	
Blood 4 c.c.	26½ Clotted as a jelly
<i>Tube C.</i> —Saline 5 c.c.	
Blood 4 c.c.	16½

EXPERIMENT 7. (Juice collected in this case was from a gastro-enterostomy and contained bile.)

<i>Tube A.</i> —Saline 3 c.c.	
Blood 3 c.c.	8½
<i>Tube B.</i> —	
Gastric juice, 0.18% HCl, 1½ c.c.	
Saliva 1½ c.c.	
Blood 2 c.c.	34 Clotted as a jelly
<i>Tube C.</i> —	
Gastric juice, 0.18% HCl, 3 c.c. . .	{ No evidence of clot at one hour
Blood 3 c.c.	

These figures appear to show that the coagulation time of blood is markedly delayed by gastric juice, and that this delayed period is shortened by the presence of saliva. It is not, however, as short as in the case of saliva

alone, even allowing for dilution, as is shown in *Experiment 3*, where dilution of 1-4 makes no difference to the clotting action of saliva.

EXPERIMENT 8.

	TIME TAKEN TO CLOT in minutes
<i>Tube A.</i> —Gastric juice, 0.2% HCl, 2 c.c. Sod. bicarb., 1-16, 2 c.c. Blood 4 c.c.	66½
<i>Tube B.</i> —Gastric juice, 0.2% HCl, 2 c.c. Saline 2 c.c. Blood 4 c.c.	Still liquid at 100 minutes
<i>Tube C.</i> —Gastric juice, no free HCl, 2 c.c. Saline 2 c.c. Blood 4 c.c.	19
<i>Tube D.</i> —Saline 4 c.c. Blood 4 c.c.	55

In this experiment the HCl in the gastric juice was neutralized by soda bicarbonate, and it will be seen that in this case the time taken to clot was longer than in the case of the gastric juice with no free HCl; also that the time taken to clot in *Tube C* was actually less than in the case of blood and saline alone. This may be accounted for by the swallowed saliva.

CONCLUSIONS.

The conclusions afforded by these experiments seem to be as follows :—
(1) That there is present in saliva a substance that hastens the clotting of blood; (2) That gastric juice with free hydrochloric acid delays the clotting of blood; (3) This delay in clotting is most marked in the presence of high acid juices such as occur in gastric and duodenal ulcers; (4) The clot formed in the presence of free hydrochloric acid is jelly-like in character and never firm.

It may be deduced therefore that the substance which hastens the clotting of blood is either destroyed or precipitated by weak acids. I would suggest that this substance is identical with tissue fibrinogen, although I have not been able to isolate it from saliva.

In a case of hæmatemesis the quantity of blood shed is frequently large and appears to be proportionately larger than that shed from extensive wounds elsewhere—say of the limbs. This cannot be entirely accounted for by the hæmostatic measures that are taken in such wounds. Further, the vomited blood seldom contains well-formed clots; it tends to be much more liquid in character.

Hæmatemesis of a severe variety is chiefly associated with simple gastric and duodenal ulcers—that is, with conditions where the acidity of the gastric juice is high. In the other common ulcer of the stomach—carcinomatous ulcer—large hæmatemesis practically never occurs, and it is to be noted that in this case the gastric juice has no free hydrochloric acid.

I would therefore deduce from these facts, and the foregoing experiments, that in a case of hæmorrhage from a simple gastric or duodenal ulcer the shed tissue fibrinogen is wholly, or in part, rendered inactive by the hydrochloric

acid, and that this necessary adjuvant to the clotting of blood being present in inadequate amount, there is marked delay in clotting, and the clots when formed are poor and jelly-like in character. Such a jelly-clot has obviously less chance of blocking an open vessel, and is very easily dislodged by the movement of the stomach.

The addition of more tissue fibrinogen to the stomach under such conditions should materially aid the clotting, and this is supplied by saliva. On the other hand, some of the extra tissue fibrinogen so supplied will be thrown out of action by the acid gastric juice, but not all, as is seen in *Experiments 5 and 7*.

Such being the physiological state, the treatment of hæmatemesis from simple gastric and duodenal ulcers would seem to be as follows:—

1. The flow of saliva should be augmented by the sucking or chewing of some innocuous substance.
2. The gastric juice should be neutralized by the frequent administration of small quantities of alkali.
3. The movement of the stomach should be restricted by rest and the administration of morphia.

PRIMARY THROMBOSIS OF THE AXILLARY VEIN: A STUDY OF EIGHT CASES.

By ERIC PEARCE GOULD,

SURGEON TO OUT-PATIENTS AT THE MIDDLESEX HOSPITAL, LONDON;

AND D. H. PATEY,

BRITISH ASSOCIATION OF SURGEONS' SCHOLAR.

PRIMARY or idiopathic thrombosis of the axillary vein is a somewhat rare, though well recognized, condition. Cadenat,¹ writing in 1920, was able to collect 27 cases, giving the credit for reporting the first case to von Schrötter in 1884. In England the condition does not seem to have attracted much attention, and the only contribution on the subject we have been able to trace is an article by Willan² recording three cases. In France, under the title of 'thrombite par effort', the clinical features have been well established, and Pellet,³ Lahaussais,⁴ Fievez,⁵ Bazy,⁶ and Hartmann⁷ have recorded examples. The German literature contains articles by Finkelstein,⁸ Rosenthal,⁹ Winterstein,¹⁰ Holländer,¹¹ and Schepelmann,¹² whilst the most notable contribution from America is by Lowenstein,¹³ who reports one case, and studies at some length the anatomical factors responsible for the lesion. As the number of cases reported by each observer has been uniformly small, and we have been fortunate enough to see or have access to the notes of eight patients, seven of whom we have been able to trace up to the present time, it was thought that a somewhat full report of the cases and a reconsideration of the etiological factors would be of interest.

Case 1.—R. Y., male, age 24, undergraduate.

HISTORY.—Three weeks before admission the patient noticed pain in the left axilla and a numb feeling at the elbow, starting immediately after an unfortunate round of golf in which he got into several bunkers. The next day, whilst playing golf again, pain in the arm became extreme and he found it swollen, with loss of movement in the fingers.

ON EXAMINATION (Aug. 10, 1914).—The patient's general condition was good; pulse, temperature, and respiration normal; the whole of the left upper arm was swollen, but did not pit on pressure. Two masses could be felt in the outer wall of the axilla corresponding to the position of the axillary vessels. A thrombus could also be felt in the basilic vein. There was some tenderness in the infraclavicular region. X-ray examination showed no abnormality in the region of the shoulder.

TREATMENT AND PROGRESS.—He was treated by rest, the swelling gradually subsided, and he left hospital on Sept. 9. Reports in December, 1927, that he had no complaint to make about the arm.

Case 2.—E. M., age 28, motor driver.

HISTORY.—Seven days before admission the patient noticed pain in the right shoulder region and swelling of the right arm. No note was taken of what the patient was doing before and at the time this occurred.

ON EXAMINATION (March 15, 1920).—His general condition was good; pulse, temperature, and respiration normal. The whole of the right upper limb was swollen and cedematous; a bluish venous patch was noticed over the shoulder, and there

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was slight fullness in the right supraclavicular fossa; Wassermann reaction negative.

TREATMENT AND PROGRESS.—Under treatment the swelling slightly diminished, especially in the forearm, but there was still swelling of the upper arm. He left hospital on April 18. We have been unable to trace his subsequent progress.

Case 3.—H. F., age 24, tailor's cutter.

HISTORY.—Fourteen days before admission the patient had a sore throat, and discharge from the right ear. He stated that at the onset of the original attack he was using an electrical cutting machine. Two days later the right arm became swollen, bluish, and painful.

ON EXAMINATION (Jan. 12, 1922).—Temperature and pulse were normal, general condition was fair, the whole of the right upper arm swollen and cyanosed. A thrombus could be felt in the basilic vein, presumably an extension. The tonsils were found to be septic, and removal of these was therefore advised.

TREATMENT AND PROGRESS.—The arm was kept at rest. The tonsils were removed. There is no further notice of the progress of the arm whilst in hospital, but his reply to an inquiry in November, 1927, was as follows: "Going on quite well, arm a little swollen after exertion. No stiffness of the joints." Is able to do his normal work.

Case 4.—C. A., age 40, labourer.

HISTORY.—Five days before admission he was working with a hammer, when he noticed the fingers of the right hand swelling. Later the hand became weak, and he was unable to grip his hammer. Gradually the whole hand, forearm, and arm became extremely painful.

ON EXAMINATION (Feb. 12, 1923).—The whole of the right upper limb was very swollen, the increased circumference as compared with the sound side varying from $1\frac{3}{4}$ in. at the wrist to $2\frac{1}{2}$ in. in the upper arm. There was tenderness over the outer wall of the axilla in the position of the axillary vein. The superficial veins running from the upper arm to the chest wall were dilated and prominent. Temperature, pulse, and respiration were normal; no evidence of infective focus.

PROGRESS AND TREATMENT.—The patient was treated by rest and elevation of the limb, followed by massage; the swelling of the arm greatly diminished. On discharge from hospital on March 6 there was still occasional swelling of the limb and some residual stiffness of the joints. On examination in November, 1927, the arm was normal in size, but there was a well-marked plexus of dilated superficial veins on the anterior aspect of the shoulder and pectoral regions. The patient was using his right arm for all his usual heavy work, but there was occasional swelling at the end of a hard day, and tasks which involved raising the arm above the shoulder level still caused an aching pain.

Case 5.—A. H. W., age 38, L.C.C. inspector.

HISTORY.—A few days before coming under observation he was assisting his wife to hang new curtain rods, which involved putting in eighteen screws above the level of his head. On the same day he noticed discoloration of the right hand and swelling of the arm, with stiffness and pain in the armpit, which got gradually worse.

EXAMINATION AND PROGRESS.—The general condition on May 21, 1923, was good, but the whole right hand was blue and swollen, with marked superficial and deep œdema of the whole arm. Dilated superficial veins were obvious over the shoulder. The axillary vein could be felt as a firm tender cord from its commencement to the lower border of the clavicle. Treatment consisted in complete rest of the arm for fourteen days, followed by commencing re-use, and a few months later vigorous exercises to restore the movements of the shoulder. When seen again on Jan. 11, 1928, there was the merest trace of swelling of the right hand, with a tendency to blueness in cold weather. The superficial veins over the shoulder, which had increased in size during the first six weeks of the illness, had disappeared and were scarcely more noticeable than those of the left. The axillary vein could no longer be felt. The patient is making full use of his right arm, and is not conscious of any difference between the two.

Case 6.—G. A., age 28, hairdresser.

HISTORY.—On the morning of admission, after placing some jars on a shelf above his head, the patient noticed that the veins of his right arm were prominent and the whole limb was swollen. Later it became painful, and he came to hospital and was admitted.

ON EXAMINATION (June 18, 1927).—His general condition was good, temperature 100°, pulse 104. The whole of the right arm was swollen and darker than the left, and a plexus of dilated veins was present in the shoulder region.

TREATMENT AND PROGRESS.—The patient was treated by rest and elevation of the limb. The swelling gradually subsided, and he was discharged on July 4, 1927. His reply to an inquiry in November, 1927, was that he was quite well, the arm swelled a little, but not enough to interfere with his work.

Case 7.—W. M., age 27, porter.

HISTORY.—On the day previous to admission, whilst cleaning a ceiling, he felt pain in the right shoulder. Later the right arm began to swell and the following day the swelling was greater, and he was admitted to hospital.

ON EXAMINATION.—The patient was a healthy muscular man; temperature, pulse, and respiration normal. The whole of the right upper extremity was very swollen and of a cyanotic tinge, but did not pit on pressure. A plexus of dilated veins was apparent on the upper part of the shoulder, the direction of the blood-flow being from the shoulder to the chest.

TREATMENT AND PROGRESS.—The patient was treated by rest and elevation of the arm, and iodoform applications. The swelling gradually subsided. A blood culture was sterile and the Wassermann reaction negative. In reply to inquiry in November, 1927, he stated that the arm was going on well, still a little bigger than the opposite side. He was doing light work, but had some stiffness on raising his right shoulder to wash his face. When examined in November some swelling of the arm could still be detected and the veins in the shoulder region were prominent. Movements were good.

Case 8.—J. P., age 23, plumber's mate.

HISTORY.—A few days after receiving some small abrasions on the right hand, while performing heavy work, he noticed pain in the right axilla, followed by swelling of the arm.

ON EXAMINATION (June 28, 1927), fourteen days after the above, a thrombosed vein was detected in the right arm, running from axilla to elbow, with considerable swelling of the arm. His general condition was good.

PROGRESS AND TREATMENT.—Treatment was by rest and elevation of the arm. When seen in December, 1927, the arm had returned to normal; the superficial veins over the right shoulder were slightly more obvious than over the left. He stated that he was using his arm for his ordinary work without disability.

Analysis of Cases.—The following are the main facts which emerge from an analysis of these cases:—

Sex.—All patients were of the male sex.

Age.—The youngest was 23 years, the oldest 40. Six out of the eight were between the ages of 20 and 30 years.

Side.—The right arm was affected in all patients except one.

General Health.—The patients were all healthy active individuals at the time of the onset of the trouble.

Apparent Cause.—In all cases in which this could be determined, the onset appeared to have been related to some muscular effort or strain.

Radiographs of the shoulder, blood culture, and the Wassermann test were negative in the instances in which these investigations were carried out.

Typical Clinical History.—There is a remarkable similarity between all the cases, and a typical history would run as follows: A young man, a few

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hours after some unusual exercise with the right arm involving work with the arm elevated, develops pain or swelling or both in the arm, increasing in the course of the next day or two until the whole arm is swollen and somewhat cyanosed. On examination there is slight, if any, elevation of temperature, and the general condition of the patient is good. The whole arm is the seat of solid swelling, with obviously dilated superficial veins running from the upper arm to the pectoral region; in the axilla a firm cord is to be felt corresponding in position to the axillary vein and possibly traceable down the arm to the elbow. Under the influence of rest, with elevation of the arm, the swelling slowly subsides, and in the course of a few weeks movements of the joints, at first limited, become again free. Improvement continues for a period of months, at the end of which time the function of the arm is fully restored as regards all ordinary uses, the only permanent evidence of the condition being some slight dilatation of the superficial veins on the shoulder and possibly some pain on full elevation of the arm.

Comparison with Recorded Cases.—Such a history appears to agree in the main with most of the recorded cases. Thus Willan reports three cases in men of ages 22, 21, 32, and all on the right side; Pellot described one case and refers to another twelve, and states that nine out of ten are right-sided, and in the male sex. Finkelstein describes two cases, one a male of 33 with the right arm affected, the other a woman of 24 in which the attack was left-sided, whilst Rosenthal reports two cases and reviews three others, all of which were right-sided. One of his cases is of special interest owing to the age of the patient, a girl 11 years old in whom the lesion followed a strain while playing at ball. Cadenat comments on the frequency of right-sided involvement. Bazy's patient was a man of 27 in whom the right arm was involved.

Winterstein described four cases, three of them males and one a female. Two of them suffered on the right side, and two on the left; but of the two latter, one was a weaver, whose work involved strain of the left arm, and the other, who was engaged in lifting bales of cloth and other heavy work, was in the habit of putting most strain on the left arm. In discussing this point Winterstein quotes Schepelmann, who reported the case of a man in whom the attack followed the riding of a restive horse. The man was naturally right-handed, but had used his left arm for controlling the horse. In most of the cases reported in which the attack was left-sided, the history reveals some definite reason for this, as in the three cases discussed by Winterstein; and in *Case 1* of our series it is probable that the efforts required to get out of a number of bunkers in a round of golf threw an extra strain on the left arm.

Pathology.—The majority of authors are agreed as to the importance of trauma in the etiology of the condition, though Wilson¹⁴ records two cases, in one of which he considered tuberculosis, and in the other syphilis, the causative factor: and in Holländer's patient there was no known injury. The history of onset, together with the fact that most of the cases are males affected on the right side, favours a traumatic origin. Moreover, the story of special left-sided strain in the patients in whom the left arm was affected affords further support. In many instances, too, the patient tells of some unaccustomed work with the right arm. In *Case 5*, for example, the attack followed

the use by a man of clerical occupation of a screw-driver above the level of the shoulder, and in *Case 7* the spending of a day cleaning ceilings by a workman accustomed to cleaning windows. Although Rosenthal suggested that the underlying lesion was a rupture of the intima, and Cadenat postulated two factors, firstly an expiratory effort distending the vein, and secondly an injury to the venous lining, to Lowenstein belongs the credit for the first serious anatomical study. By means of dissections he demonstrated that, in certain individuals, in the abducted position of the arm the costocoracoid ligament makes a definite indentation in the axillary vein. He therefore suggested that this structure is responsible for the trauma to the vein when the arm is abducted. His studies also led him to deny that pressure from the head of the humerus was of importance, as believed by Willan.

We have endeavoured to throw light on the matter by injecting plaster-of-Paris into the axillary veins of post-mortem subjects, the arms of which were held fixed in the abducted position. In one instance a shallow groove was present in the cast so obtained corresponding to the position of the costocoracoid ligament, while in two other casts a broad deep groove from the pressure of the subclavius muscle was seen. In addition, numerous dissections showed that a practically constant anatomical feature of the vein at the latter level was a competent bicuspid valve, so placed that pressure by the subclavius muscle stretched the vein wall in the long axis of the valve. The valve cusps are very delicate structures, composed only of a thin sheet of fibrous tissue covered on either side with endothelium. We are therefore of the opinion that the subclavius muscle is responsible for the trauma to the vein, and suggest that a rupture of this delicate subclavio-axillary valve is the fundamental pathological lesion. As predisposing factors there are, as previously quoted, a distended condition of the vein due to expiratory effort, and the abducted position of the arm.

Differential Diagnosis.—The history and clinical features are usually so typical as to allow of diagnosis at a glance. In doubtful cases, the possibility of syphilis and tuberculosis should be considered. A skiagram will show any bony abnormality, or an intrathoracic tumour, whilst the thrombosis of the subclavian and axillary veins sometimes met with in cardiac failure is easily distinguished by the general condition of the patient. We have met with one case in which the diagnosis of primary axillary thrombosis was wrongly made—a man, age 43, in whom the left arm was affected. The condition subsided, and he was able to rejoin the army, but eight months later it was discovered that he had an aneurysm of the arch of the aorta. Before death the arm again became very swollen.

Prognosis and Treatment.—The prognosis appears to be uniformly good. All that is required in the way of treatment is elevation of the arm for two to three weeks, followed by massage. Embolism is a theoretical danger, but in fact the only case in which this has occurred is one (quoted by Cadenat) in which Schepelmann operated and removed the clot. Bazy and Guyot, and Jeanneney,¹⁵ have also operated upon the vein. Wilson mentions one case which was treated by multiple incisions to relieve œdema. It would appear that all such drastic active methods are unjustifiable.

SUMMARY.

Detailed notes are given of eight cases of primary thrombosis of the axillary vein.

From these a typical history is constructed, and this is compared with those of cases previously published. The etiology is discussed, and short comments are added upon the differential diagnosis, treatment, and prognosis.

REFERENCES.

- ¹ CADENAT, F. M., *Paris méd.*, 1920, i, 253.
- ² WILLAN, R. J., *Edin. Med. Jour.*, 1918, xx, 105.
- ³ PELLOT, *Presse méd.*, 1916, xxiv, 523.
- ⁴ LAHAUSOIS, M., *Ibid.*, 1910, xviii, 410.
- ⁵ FIEVEZ, *Bull. Mém. Soc. de Chir. de Paris*, 1926, lii, 529.
- ⁶ BAZY, L., *Ibid.*
- ⁷ HARTMANN, *Rev. gén. de Clin. et de Thérap.*, 1927, xli, 70.
- ⁸ FINKELSTEIN, B., *Deut. med. Woch.*, 1927, Jan., 198.
- ⁹ ROSENTHAL, W. J., *Deut. Zeits. f. Chir.*, 1912, cxvii, 405.
- ¹⁰ WINTERSTEIN, O., *Schweiz. med. Woch.*, 1925, April, 360.
- ¹¹ HOLLÄNDER, E., *Berl. klin. Woch.*, 1921, lviii, 182.
- ¹² SCHEPELMANN, *Behefte z. med. Klin.*, 1911, vii, 23.
- ¹³ LOWENSTEIN, P. S., *Jour. Amer. Med. Assoc.*, 1924, March 15, 854.
- ¹⁴ GUYOT and JEANNENEY, *Bull. et Mém. Soc. de Chir. de Paris*, 1923, xlix, 231.
- ¹⁵ WILSON, G., *Amer. Jour. Med. Sci.*, 1922, June, 899.

THE SIGNIFICANCE OF HEPATITIS IN RELATION TO CHOLECYSTITIS: AN EXPERIMENTAL STUDY.*

By A. L. WILKIE, MONTREAL.

IN recent discussions on gall-bladder infections the relation of the so-called hepatitis to cholecystitis has occupied a great deal of attention. Based on Graham's¹ observations, the theory of a primary hepatitis with a secondary lymphatic spread of the infection to the gall-bladder claims many supporters. The presence of pathological liver changes in conjunction with gall-bladder inflammatory disease has long been recognized, and so the question arose, and is still a matter of dispute, whether the hepatitis or the cholecystitis is the primary lesion. In a previous communication³ it was stated that in experimental animals, where cholecystitis had been produced by direct inoculation of bacteria into the wall of the gall-bladder, it was possible to trace the spread of infection to the surrounding liver substance. This fact was clearly observed by means of exploratory laparotomies from time to time. In order, however, to provide more conclusive proof that the gall-bladder is the seat of the primary lesion and the liver changes are secondary, the following series of experiments was carried out.

TECHNIQUE OF THE EXPERIMENTS.

In these experiments rabbits only were employed. The abdomen was opened, and the gall-bladder dissected and completely separated from its liver-bed, its only connection remaining being the cystic duct and the cystic vessels. A small portion of omentum was then interposed between the liver and the gall-bladder, and the latter drawn back into position by means of suture. In several of the animals the cystic duct was ligated, great care being taken not to include the vessels. In this way any lymphatic or blood connection that might exist between the gall-bladder and its bed was completely severed, and the interposed omentum served as a barrier between the two. About three weeks were allowed to elapse before any further procedure was adopted, so that all animals might completely recover from the primary operation. At the end of four months animals which were used as controls showed no ill-effects from this operation, and when killed the gall-bladder showed no definite pathological change. The interposed omental tissue remained and the neighbouring liver substance was normal (*Fig. 193*). The microscopie study was carried out by means of complete transverse sections through the gall-bladder and neighbouring liver substance.

The experiments on the remaining animals, prepared as already stated,

* From the Department of Experimental Surgery, University of Edinburgh: Director Professor D. P. D. Wilkie.

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may be classified under two headings: (*Group 1*) The production of cholecystitis by intravenous injections of streptococci in those animals where the cystic duct had been previously ligated; (*Group 2*) The production of cholecystitis by intravenous injections of streptococci in the animals in which the cystic duct was not occluded.

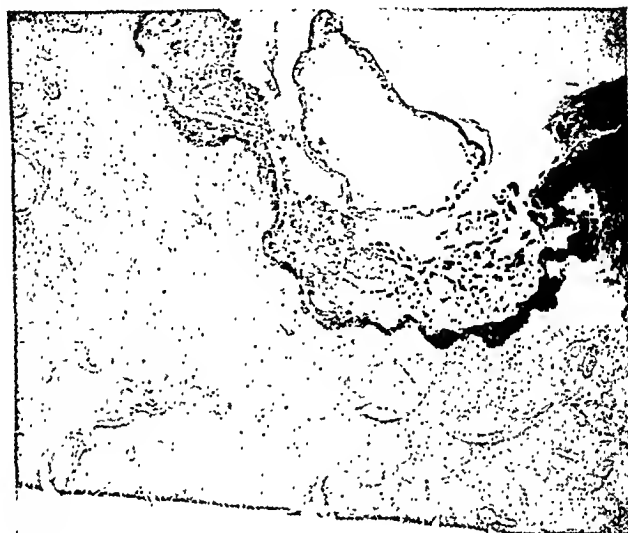


FIG. 193.—Transverse section of rat liver-bed, showing omentum interposed, four months after operation on gall-bladder walls.

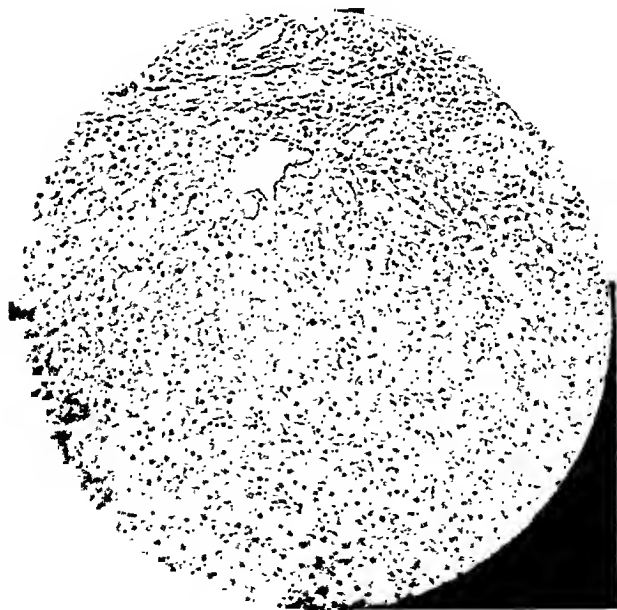


FIG. 194.—High-power photograph of liver substance at the margin of interposed omentum. Note the absence of any definite inflammatory change in the liver substance itself.

Group 1.—In this group it is fairly evident that, since the gall-bladder was separated from its liver-bed and the cystic duct tied, organisms could be conveyed to the gall-bladder only by means of the blood-stream through the cystic artery. The organisms injected were pure cultures of streptococci in saline suspension previously isolated from human cystic lymph glands draining diseased gall-bladders. These streptococci, when injected intravenously in small numbers at weekly intervals, have been shown to produce a slow but progressive cholecystitis in animals.^{2,3} The animals in this group of experiments received from six to seven intravenous inoculations at weekly intervals, and were killed at the end of four months. In all cases a definite chronic



FIG. 195.—Rabbit's gall-bladder and liver four months after intravenous streptococcal injections. Note omentum interposed between gall-bladder and liver. Marked chronic cholecystitis. (Inset: Calculus composed mainly of cholesterol removed from the interior of this gall-bladder.) (Actual size.)

cholecystitis was observed, characterized by marked thickening of the gall-bladder wall, due to inflammatory fibrous tissue associated with small-celled infiltration. In the region of the liver-bed the interposed omentum had fused with the gall-bladder wall, making it very difficult to distinguish the line of demarcation. The line of demarcation of liver substance, however, was quite sharp. The liver substance appeared normal, and no evidences of any inflammatory infiltration could be observed (*Fig. 194*). The liver changes so commonly associated with cholecystitis were absent.



FIG. 196.—Cholecystitis produced by means of intravenous inoculation, the gall-bladder having been previously separated from the liver-bed. Note marked inflammation of all layers, and inflammatory-celled infiltration. This is well seen in the



FIG. 197.—Microphotograph of normal gall-bladder of rabbit, photographed with the same power as Fig. 196 for comparison.

Group 2.—In the second group of experiments a technique similar to that of *Group 1* was employed, with the exception that the cystic duct was not ligated. After repeated intravenous streptococcal injections, cholecystitis developed as before (*Fig. 195*). Microscopic sections showed an enormous increase of inflammatory tissue associated with cellular infiltration in all coats (*Figs. 196, 198*). This is well seen when compared with the rabbit's normal gall-bladder photographed with the same power (*Fig. 197*). The demarcation of liver substance, as before, was clear. No inflammatory infiltration was observed in spite of the great reaction in the gall-bladder wall itself (*Fig. 199*).

Finally, as a control, organisms from the same cultures as those used above were injected intravenously into rabbits without any previous operative procedure. Cholecystitis developed as already recorded, and when a period of four months had elapsed transverse sections of the gall-bladder and liver-bed were made. The gall-bladders showed typical intramural chronic



FIG. 198.—High-power photograph of a portion of *Fig. 196*.

(productive) changes. The inflammatory reaction, however, was not confined solely to the gall-bladder wall, but had invaded the surrounding liver substance as well (*Fig. 200*). The line of demarcation between the liver substance and the gall-bladder was ill-defined. The liver cells themselves were pale and hazy, inflammatory-celled infiltration was present, and an increase of fibrous tissue observed in the interlobular areas, especially surrounding small bile channels. This picture was a contrast to those of the former groups of experiments.



FIG. 199.—Section of rabbit's liver at the margin of interposed omentum. Note sharp line of demarcation of liver substance, showing no definite inflammatory reaction.



FIG. 200.—Section of rabbit's liver taken at a short distance from the gall-bladder bed. This animal received five weekly intravenous streptococcal inoculations. No previous operation had been carried out. Note inflammatory processes invading liver substance in irregular patches.

COMMENTARY.

From these simple observations certain facts may be deduced. Cholecystitis produced in previously normal animals by the intravenous injection of streptococci was also seen following the injection of similar organisms into animals in which the gall-bladder had previously been separated from its liver-bed. Even when the cystic duct in addition was occluded, chronic intramural cholecystic changes resulted from intravenous inoculations of streptococci. The organism, therefore, must have reached the gall-bladder by the cystic artery. Further, in the cases where the gall-bladder was separated from the liver, and cholecystitis produced, no hepatitis resulted. In cases where the gall-bladder was undisturbed, hepatitis was invariably present. Therefore it seems logical to assume that the liver changes produced in the latter group were the result of a direct spread of the infection from the *primary* gall-bladder lesion. Where lymphatics and blood-vessels, if present, were severed, and omentum interposed between the gall-bladder and its liver-bed, the routes of spread were cut off and no hepatitis resulted.

CONCLUSIONS.

1. There is experimental evidence that the intramural gall-bladder lesion in cholecystitis precedes the common liver changes in that disease.
2. The infecting organism in all probability reaches the gall-bladder wall by way of the blood-stream.

In connection with this work I wish to express my thanks to Professor D. P. D. Wilkie and to the technical staff of the Department of Experimental Surgery, University of Edinburgh.

REFERENCES.

-
- ¹ GRAHAM, E. A., "Further Observations on the Lymphatic Origin of Cholecystitis, Choledochitis, and the associated Pancreatitis", *Arch. of Surg.*, 1922, Jan., iv, 23.
 - ² ROSENOW, E. C., "Bacteriology of Cholecystitis and its Production by Injection of Streptococci", *Jour. Amer. Med. Assoc.*, 1914, Nov. 21, 1835, and other papers.
 - ³ WILKIE, A. L., "The Bacteriology of Cholecystitis", *Brit. Jour. Surg.*, 1928, xv, 450.

GANGLIONEUROMATOSIS OF THE ALIMENTARY TRACT.

BY HUGH POATE AND KEITH INGLIS, SYDNEY.

CLINICAL REPORT (HUGH POATE).

EX-PRIVATE B. T., 30 years of age, was admitted to hospital in April, 1925, complaining of flatulence, fullness and discomfort in the epigastrium, and pain behind the right shoulder. The symptoms were most noticeable just before meals or when the patient was in a cramped position for long—for example, whilst riding or driving. There had been some fullness and pain in the right side of the abdomen; the pain was worse if he was constipated. From 1923 onwards the bowels had tended to become sluggish. Symptoms had been complained of since 1919, but the dyspepsia had been worse during the nine months immediately preceding the last operation. About twelve months prior to this operation there had been some dry retching, but there had never been any vomiting or mæna. In 1919 the patient's weight was 11 st. 8 lb., but at the time of his second operation it had fallen to 10 st. 4 lb. He was blown up at Messines in June, 1917; he sustained no obvious injury, but while in hospital in England it was noticed that he had a swelling in the right loin. An operation was performed, and it is stated in his record that "a cystic retroperitoneal tumour was removed from the right loin. The kidney was found to be normal".

PHYSICAL EXAMINATION.—This revealed nothing abnormal except a peculiar fullness in the right side of the abdomen. There was no tenderness. Opaque-meal examinations with the X rays showed a constant deformity of the duodenal cap, with a reflex transient incisura near the fundus of the stomach. The X-ray diagnosis was chronic duodenal ulcer. No examination of the intestinal tract was made.

OPERATION (May 7, 1925).—The abdomen was opened by a right supra-umbilical paramedian incision. A chronic ulcer was found on the anterior wall of the first part of the duodenum, and a large soft mass was felt in the ascending colon. This mass, which resembled a chronic intussusception extending as far as the hepatic flexure of the colon, was composed partly of a soft neoplasm inside the bowel, and partly of cæcum and ascending colon which were greatly distended by the growth. The whole mass was delivered through the incision without any difficulty, and the portion of bowel between the terminal ileum and proximal part of the transverse colon was resected. The ends of the bowel were closed, and a lateral anastomosis between ileum and transverse colon was performed. All raw areas were covered with peritonium. The duodenal ulcer was then oversewn and a posterior gastrojejunostomy performed.

The patient showed no sign of shock and made an uninterrupted recovery. When interviewed some eighteen months later he was free from all symptoms and showed no evidence of any abnormal state.

PATHOLOGICAL REPORT (KEITH INGLIS).

Macroscopic Examination.—The specimen (*Fig. 201*) was examined after fixation in Kaiserling's No. 1 solution (10 per cent formalin) and mounting in Frost's (sugar) solution. It consists of the lowest portion (4·5 cm. long) of the ileum, the cæcum, the appendix, and the ascending colon, all in one piece. The external surface of all portions of the specimen presents no abnormality.

The Ileum.—Projecting into the lumen of the ileum are two firm nodules; the larger is situated 4 cm. from the ileocæcal junction, is approximately 1 cm. in diameter, and is attached to the wall of the bowel by a flaccid pedicle approximately 1 cm. long; the smaller is situated 3 cm. from the

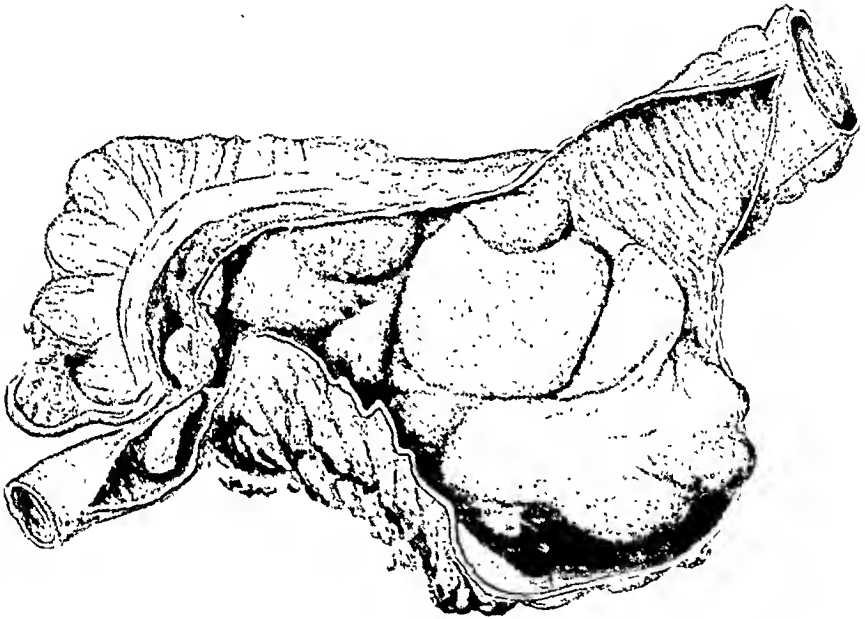


FIG. 201.—Specimen removed at operation.

ileocæcal junction, is 0·8 cm. in diameter, and has practically no pedicle. Both nodules are covered by apparently intact mucous membrane.

The Cæcum and Appendix.—In the cæcum are two irregularly rounded tumour masses projecting into the lumen; the larger is approximately 4 cm. in diameter, situated in the fundus inferior to the ileocæcal junction; the smaller is approximately 1·5 cm. in diameter, and is situated at the junction of the cæcum with the ascending colon. Apart from these two isolated tumours, quite two-thirds of the wall of the cæcum is diffusely thickened by neoplastic tissue. The thickened portion measures approximately 1 cm. from scrous to mucous surface, whereas the normal portion is only a few millimetres thick. The appendix, which is 10 cm. long and 0·5 cm. in diameter, was not examined microscopically. To the naked eye it presents no features of special significance.

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The Ascending Colon.—The main growth commences 1 cm. above the ileocaecal junction and extends approximately 20 cm. up the ascending colon; about nine-tenths of this portion of the bowel is involved in new growth, which takes the form of lobulated tumours projecting into the lumen. The greater part of the growth is composed of three large masses each measuring approximately 10 cm. by 5 cm. and projecting 3 to 4 cm. into the lumen. The margins of these masses are overhanging, and the surfaces, though slightly irregular, are smooth and covered by apparently intact mucosa. The contour of these masses may have been altered during fixation. In addition to these three main tumour masses there are some smaller ones which, except for size, are almost identical with the larger ones. From an examination of the

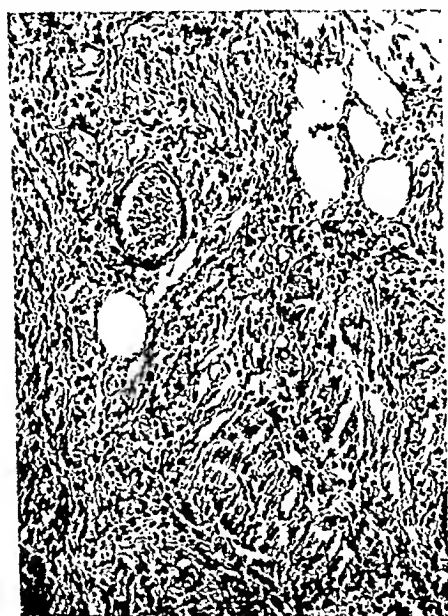


FIG. 202.—Illustrates the fibrocellular structure of which the greater part of the tumour is composed. A small amount of adipose tissue is enmeshed in the growth. ($\times 100$.)



FIG. 203.—Showing abnormal nerve-trunks, a form assumed by the growth in places. Intermediate stages between those illustrated in Fig. 202 and Fig. 203 occur. ($\times 48$.)

specimen it seems likely that, apart from the tumour masses, the ascending colon is somewhat larger than normal, but this is uncertain.

Microscopic Examination.—Both large and small tumours were examined microscopically, and the structure is much the same in both. The greater part of the tumour masses is composed of fibrocellular tissue (Fig. 202) in which little resemblance to nerves can be detected. In places the tumour is composed mainly of enlarged and abnormal nerve-trunks (Fig. 203), and intermediate stages between these two extremes are to be seen. Some of the abnormal trunks are surrounded by dense fibrous tissue. These fibrous zones may be the result of long-standing inflammation, but I think it is more probable that they are part of the new growth.

The tumour masses are situated within the circular muscle of the bowel

in the region of Meissner's plexus. Though most of the neoplastic tissue is external to the muscularis mucosæ, growth is also present to a slight extent



FIG. 204.—Includes a cluster of ganglion cells situated in the substance of a small tumour mass which was approximately 0.5 cm. in diameter. ($\times 175$.)



FIG. 205.—Showing nerve-cells (mostly degenerate) within and without the muscularis mucosæ. The portion of tissue included in this illustration was situated on the surface of one of the larger tumour masses. ($\times 95$.)



FIG. 206.—Photomicrograph taken from the end of a section. It includes a portion of an abnormal vessel. The tissue accounting for the increased thickness of the vessel wall is possibly of neoplastic and not inflammatory origin. ($\times 50$.)

within the muscularis. Ganglion cells are evident in large numbers, sometimes scattered widely, the individual nerve-cells being isolated, at others collected into clusters. The great majority of the ganglion cells are degenerate, but here and there they are sufficiently well preserved to place their identity beyond doubt. The degenerate changes in the nerve-cells may be accounted for in part by bad fixation, but in sections from other specimens treated in the same way as this one, the ganglion cells are much more distinct, so that probably faulty fixation is not entirely to blame for the degeneration of the nerve-cells in this tumour. *Fig. 204* shows a cluster of nerve-cells in one of the tumour nodules. *Fig. 205* shows nerve-cells (mainly degenerate) within and without the muscularis mucosæ. The surface epithelium in the mucous

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membrane appears fragmentary; this may be due partly to the fact that the specimen was packed with cotton-wool, but it may be due also to the inflammatory reaction which is very conspicuous in the mucous membrane. A striking feature of the inflammatory tissue is the large number of eosinophils present. Though most conspicuous in the mucous membrane, inflammatory cells, particularly eosinophils, are fairly numerous in the tumour substance proper.

In a plexiform neuroma of the hand and forearm reported by Bell and Inglis¹, the walls of some vessels of the part showed thickening of an unusual kind; this was thought to be due to involvement of the nerves of the vessel in the pathological process. In the present specimen there are some vessels with thickened walls (*Fig. 206*) in which the changes are somewhat unlike those of inflammation, and though the appearances are less striking than in the case cited, it is possible that the tissue accounting for the increased thickness of the vessel wall is neoplastic and not inflammatory. The inclusion of adipose tissue in the substance of the growth is evident in *Figs. 202 and 205*.

Until recently neurofibromata were regarded as of mesoblastic origin; but, as pointed out by Roman and Arnold² when reporting a case of ganglioneuroma of the pelvic tissue and organs, etc., in a child, recent work suggests that they are made up of nerve-fibre cells of Schwann, which are now considered to be of epiblastic origin. If these opinions are correct, probably all the neoplastic tissue in our specimen is of neurogenic origin.

All the sections examined were cut from tissue embedded in paraffin; most of them were stained with hæmatoxylin and eosin, a few of them by van Gieson's method.

REFERENCES.

¹ BELL, G., and INGLIS, K., *Med. Jour. of Australia*, 1925, Oct. 3, 423.

² ROMAN, B., and ARNOLD, D. P., "A Case of Ganglio-neuromatosis", *Bull. of Buffalo Gen. Hosp.*, 1924, July, 88.

THE SIGNIFICANCE OF THE BLOOD-CHOLESTEROL IN GENITO-URINARY SURGERY.

By JAMES MAXWELL,

PHYSICIAN TO OUT-PATIENTS, ROYAL CHEST HOSPITAL, LONDON.

THE problem of the functional efficiency of the kidneys, and the consequent ability of any given patient to combat successfully the strain of an operation upon the urinary tract, is one which has exercised the mind of surgeon and pathologist alike for many years. The apparent clinical condition of the patient is not by any means an infallible index of his powers of resistance to the additional strain thrown upon his excretory mechanism by a surgical operation, and consequently the assistance of the chemical pathologist has been invoked in order to supply additional information on this point.

The estimation of renal function by means of the blood-urea and urea-concentration tests affords information of value to the clinician, but now and again disaster occurs in spite of laboratory results which would seem to indicate a reasonable margin of safety for operative procedure. The latent factor which at times defies both clinical examination and chemical tests appears to be a lowered power of resistance on the part of the patient to the bacterial infection which is an almost invariable concomitant of operations on the lower urinary tract.

Attempts have therefore been made by many observers to estimate beforehand the importance of this factor in each individual case, and particular attention has been paid by MacAdam and Shiskin¹ to the value of estimations of the cholesterol content of the plasma from a prognostic point of view. As a result of the investigation of 88 cases of prostatic enlargement they formulated certain conclusions which may be briefly summarized as follows.

The cholesterol content of the plasma appears to be a fair measure of the degree of resistance to infection possessed by the individual, and such estimations form a useful guide to the degree of operative risk in cases of urinary obstruction. Assuming the lower limit of the plasma-cholesterol in health to be 0.130 gm. per cent—a point which will be discussed later—they found that, of 18 cases yielding a value below this limit, 16 died of pyelonephritis while only 2 recovered. Of the 70 cases yielding a cholesterol value above 0.130 gm. per cent, 11 died, only one of them, however, as a result of an ascending urinary infection. It was also noted that, in a large proportion of the cases in which a low cholesterol value was found, the clinical condition did not contra-indicate operation, and such clinical evidence as there was depended upon some degree of defect in renal function rather than upon the latent sepsis from the effects of which the patients ultimately succumbed. They considered that the test is of much less value in cases of malignant disease, the cholesterol figure being frequently high even when pyelonephritis co-exists.

Thus, from these conclusions, it would appear that we have now available

a test which will yield information of great prognostic value to the genito-urinary surgeon—a test, indeed, which is capable of correctly forecasting disaster in cases which clinically appear to be reasonably good operative risks. The value of this test therefore promises to be such that any additional cases are worthy of record, and the sole object of this paper is to report a short series of 26 cases, with a critical survey of a few of the theoretical considerations underlying the test. The cases here recorded formed part of a larger series of renal conditions of all types, the ‘medical’ cases having been already reported elsewhere.² In each case the method of estimation was identical with that employed by MacAdam and Shiskin,³ so that the results should be strictly comparable.

THE FACTORS WHICH INFLUENCE THE PLASMA-CHOLESTEROL.

At the time of the publication of the paper quoted above,¹ the normal limits for the plasma-cholesterol were thought to be quite clearly defined, and numerous references were adduced in support of figures between 0·13 and 0·19 per cent, with an average value of 0·16 per cent. More recent observations by Gardner,⁴ however, suggest that much wider limits may be compatible with perfect health, figures varying between 0·08 and 0·23 per cent having been recorded in normal adults. Thus it can be seen that a result of 0·11 per cent need not now be regarded as a finding of definite pathological significance, whereas a short time ago such a result would have been classed under the heading of hypocholesterinæmia.

It is very probable that each individual in health has a definite level, somewhere between the limits quoted, from which level there is normally but little variation. In disease, therefore, it is possible to vary from that level to quite a considerable degree before the figure transcends the so-called normal limits, and so the less gross abnormalities may pass unrecognized. Thus, in an individual whose plasma-cholesterol is normally in the region of 0·13 per cent, disease might easily cause a rise to 0·19 per cent, but this figure would have to be accepted as within the normal limits, and its proper significance would therefore not be accorded to it. Conversely, we are not strictly justified in accepting as normal a plasma-cholesterol, even within the limits quoted above, in a patient suffering from any disease, unless we have also a record of a previous finding before the onset of the disease.

In this respect it must be clearly recognized that the interpretation of plasma-cholesterol results differs considerably from that of the other common chemical examinations of the blood. The blood-urea and blood-sugar have much less variable normal values, and neither is so liable to be affected by a multitude of external factors, partly recognized, but still partly unknown, as is the plasma-cholesterol.

The following factors, singly or together, may play a part in causing changes in the plasma-cholesterol, only such factors being considered here as are likely to be of influence in genito-urinary cases:—

Age.—There is a well-known tendency for the plasma-cholesterol to be raised in association with arteriosclerosis;² therefore the average normal values in the cases under discussion, all of which were over 50 years of age,

will have a tendency to run at a higher level than would be found in a corresponding group of younger adults.

Anæmia.—In all conditions associated with a marked degree of anæmia it has been shown by MacAdam and Shiskin³ that the plasma-cholesterol is diminished, the decrease becoming marked when the red-cell count falls below 50 per cent of the normal. This factor must therefore be considered in all cases in which severe hæmaturia has occurred prior to the plasma-cholesterol determinations, and low results should be interpreted in conjunction with the red-cell count.

Infection.—In the majority of cases of disease in the lower part of the urinary tract the bladder is already infected to some extent; in the remaining cases it rapidly becomes so following operation. It is the influence of this particular factor which is the subject of the present paper, so that it would be well to consider what is already known concerning the reaction of the plasma-cholesterol to infections. In most acute infections the plasma-cholesterol is found to be low, especially in patients dying of the disease,^{5, 6} and these observations are borne out by several cases in this series. On the other hand, during recovery and the development of immunity the plasma-cholesterol is said to increase,⁶ although the mechanism by which this occurs, and its significance, are as yet imperfectly understood.

Uræmia.—In this condition normal figures are usually found,² and probably, of itself, this factor may be ignored in the interpretation of the plasma-cholesterol results.

Malignant Disease.—The results obtained in cases of malignant disease are apt to be confusing. Some authors, e.g., Luden,⁷ have reported high figures in malignant disease. Brunton,⁸ on the other hand, obtained very variable results in such cases, and my own experience has been similar. It must be remembered, also, that frequently the diagnosis of carcinoma in a prostate is only made by microscopical examination, so that, as far as interpretation of results is concerned, all cases of prostatic enlargement should be considered together; it is only rarely that one is able entirely to exclude the possibility of malignant change in a prostate, solely upon clinical evidence.

Acute Retention of Urine.—This appears at times to be an important factor in influencing the behaviour of the plasma-cholesterol, and its influence upon the findings will be discussed later.

SUMMARY OF RESULTS OBTAINED.

The cases reported here are not strictly confined to cases of prostatic enlargement. Pre-operative diagnosis is not infallible, so that it was thought preferable to take any cases of disease in the lower urinary tract and investigate the state of the blood, correlating the results obtained with the clinical, operative, and, when necessary, post-mortem findings. The case-incidence was as follows:—

Prostatic enlargement (innocent)	17
Prostatic enlargement (malignant)	2
Papilloma of bladder..	1
Carcinoma of bladder	3
Stricture with acute retention of urine	3
Total	26

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In each case several cholesterol estimations were performed at intervals, and the results were then expressed in the form of curves, several of which are appended. It was found that, by this method, more could be learned of the behaviour of the plasma-cholesterol under different circumstances; in all cases at least one observation was made before any operation was carried out.

Cases having a Low Plasma-cholesterol (Table I).—For purposes of comparison with the previously recorded cases, the value of 0.13 per cent is accepted as the dividing line, figures below this being regarded as low. As has been already pointed out, recent work suggests that this attitude is not strictly correct.

Table I.—PROSTATIC ENLARGEMENT (INNOCENT), WITH LOW PLASMA-CHOLESTEROL.

NO.	BLOOD CHOLESTEROL	BLOOD UREA	OPERATION	REMARKS
	Per cent	Per cent		
43	0.09	0.28	C	Post-mortem showed pyelonephritis
57	0.08	0.064	C + P	Recovered
69	0.12	0.035	P	Pyæmia. Died
87	0.12	0.15	C	Recovered
104	0.12	0.07	C	Recovered

C = Cystotomy only. C + P = Cystotomy with subsequent prostatectomy.
P = Single-stage prostatectomy.

Of the 17 cases of innocent enlargement of the prostate, 5 were classed in this group. Of these cases, 3 recovered and 2 died, in both of which

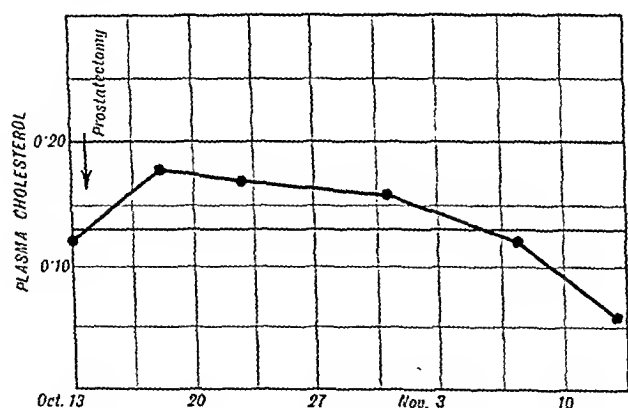


FIG. 207. —Acute retention on October 10: death from pyæmia.

pyelonephritis was found on post-mortem examination. In one of these cases, however (No. 43), a severe degree of uræmia was already present before the patient came under observation, and death appeared to be more directly due to this cause than to the concurrent infection. In the second case (No. 69) the patient was suffering from acute retention of urine with an initial plasma-cholesterol of 0.12 per cent. A single-stage prostatectomy was followed by a rise in the cholesterol to 0.18 per cent four days after operation. Ten days

later the temperature rose suddenly, with rigors and other evidence of pyæmia, and from this time the cholesterol rapidly fell until the patient's death. At post-mortem abscesses were found in the kidneys, heart, and other organs. The curve obtained from this case is set forth in *Fig. 207*. For purposes of comparison, *Case 87*, with an initial plasma-cholesterol of 0.12 per cent was investigated at intervals for six months, with results always between 0.12 and 0.15 per cent. He has subsequently undergone the second stage of the operation successfully.

Cases having a Normal Plasma-cholesterol (*Table II*).—Of the 12 cases in this group, 5 were clinically bad operative risks, in spite of the normal

Table II.—PROSTATIC ENLARGEMENT (INNOCENT), WITH NORMAL PLASMA-CHOLESTEROL.

No.	BLOOD CHOLESTEROL	BLOOD UREA	OPERATION	REMARKS
	Per cent	Per cent		
46	0.145	0.034	Nil	Clinically unfit for operation
56	0.15	0.04	P	Good recovery
63	0.20	0.08	P	Septicæmia. Death
73	0.16	0.06	C + P	Good recovery
81	0.15	0.03	C	Death following pyelonephritis
88	0.20	0.03	Nil	Clinically unfit for operation
89	0.13	0.05	Nil	Clinically unfit for operation
119	0.17	0.03	P	Good recovery
120	0.19	0.04	C + P	Good recovery
154	0.19	0.69	Catheter	Died in uræmia
155	0.13	0.21	Catheter	Discharged unfit for operation
159	0.13	0.04	C + P	Good recovery

C = Cystotomy only. C + P = Cystotomy with subsequent prostatectomy.
P = Single-stage prostatectomy.

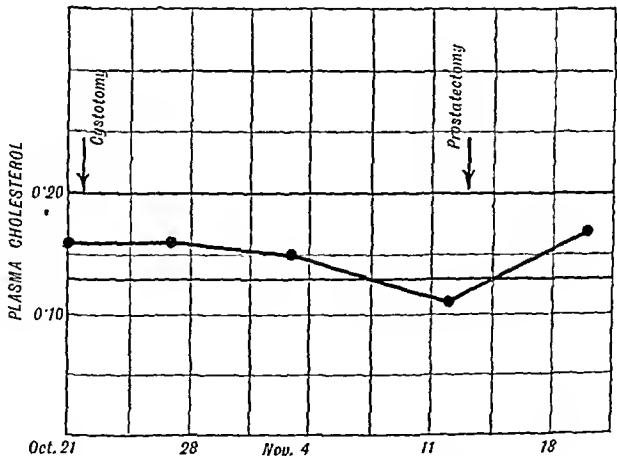


Fig. 208.—Innocent enlargement of the prostate: good recovery.

cholesterol figures, and no operation was undertaken; one of these cases (No. 154) died in uræmia very soon after admission to hospital. Of the remaining 7 cases, 5 recovered following operation and 2 developed septic

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complications and died. Thus, out of 12 cases, all showing a normal initial cholesterol figure, only 5 progressed safely through operation to recovery.

Two charts are shown from among the cases which recovered following operation. In the first case (*Fig. 208*) the two-stage operation was per-

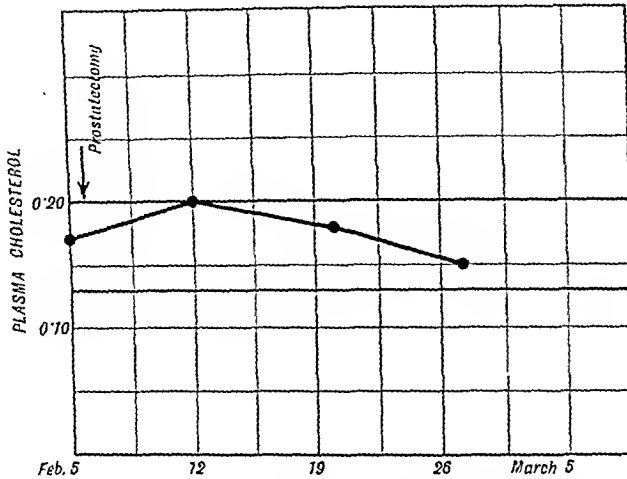


FIG. 209.—Innocent enlargement of the prostate: good recovery.

formed. The cholesterol fell following operation, and the second stage was carried out when the plasma-cholesterol was 0.11 per cent. Nevertheless the patient made a perfect recovery. The second case (*Fig. 209*) illustrates the

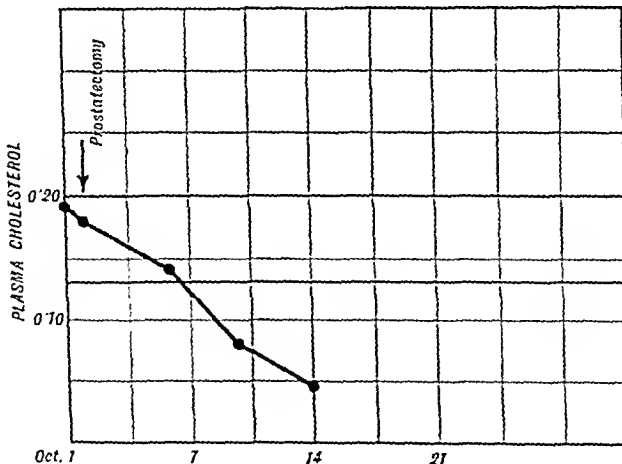


FIG. 210.—Innocent enlargement of the prostate: septicæmia, death.

changes which occurred following upon the one-stage operation, a perfect recovery again ensuing in this case. In the other cases which recovered similar curves were found. By way of contrast to the curves already described, is the chart shown in *Fig. 210*. The patient was a healthy man, age 62, in whom all the tests were favourable, and whose plasma-cholesterol

before operation was between 0.18 and 0.20 per cent. Three days after operation he developed a wound infection, rapidly followed by streptococcal septicaemia, and died on the fourteenth day after operation. The steady and rapid drop in the cholesterol content of the plasma is very striking. In the other case which terminated fatally as a result of pyelonephritis, the cholesterol fell from 0.15 to 0.09 per cent within sixteen days after operation, the type of infection being somewhat less acute than in the preceding case.

Carcinoma of the Prostate (Table III).—Only two cases of this nature were examined, the initial cholesterol findings being 0.17 and 0.12 per cent

Table III.—PROSTATIC ENLARGEMENT (MALIGNANT).

No.	BLOOD CHOLESTEROL	BLOOD UREA	OPERATION	REMARKS
163	Per cent 0.17	Per cent 0.05	C + P	Good recovery
174	0.12	0.04	C	Recovered

C = Cystotomy only. C + P = Cystotomy with subsequent prostatectomy.

respectively. Both recovered uneventfully after the operative measures indicated in the table.

Bladder Conditions (Table IV).—In three cases of carcinoma of the bladder, the findings set out in Table IV were very uniform, and the operation

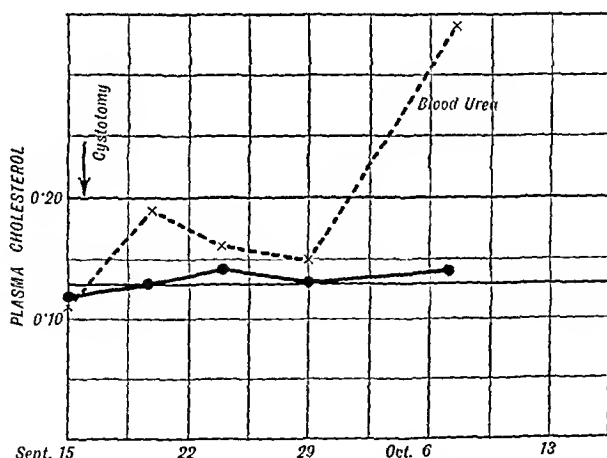


FIG. 211.—Carcinoma of bladder: death in uræmia.

of suprapubic cystotomy was performed in each. In the first pelvic peritonitis set in with fatal result, the cholesterol falling as in the other septic cases. In the second death occurred as the result of uræmia, the findings being reproduced in the chart (Fig. 211); it will be seen that the cholesterol remained very nearly constant. In the third case improvement resulted from the operation, although the growth itself proved to be inoperable.

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In a case of papilloma of the bladder the patient had previously bled profusely, exhibiting a severe degree of anæmia on coming under observation. The plasma-cholesterol was 0.16 per cent, but the patient succumbed to pneumonia a few days after suprapubic cystotomy.

Table IV.—BLADDER CONDITIONS.

NO.	CONDITION	BLOOD CHOLESTEROL	BLOOD UREA	RESULT	REMARKS
		Per cent	Per cent		
21	Carcinoma	0.12	0.03	Died	Pelvic peritonitis
59	"	0.12	0.14	Died	Uræmia
61	"	0.13	0.03	Improved	Cystotomy only
10	Papilloma	0.16	0.07	Died	Severe anæmia. Pneumonia

Acute Retention of Urine due to Urethral Stricture (Table V).—Three cases were investigated. In the first two low figures were found, and in each there was a rapid rise in the plasma-cholesterol following relief of the obstruction. In the third case the blood-urea was already 0.25 per cent, and the plasma-cholesterol 0.135 per cent on first investigation. Suprapubic cystotomy was performed, but the blood-urea continued to rise while the

Table V.—STRICTURE WITH ACUTE RETENTION.

NO.	BLOOD CHOLESTEROL	BLOOD UREA	RESULT	REMARKS
	Per cent	Per cent		
34	0.09	0.095	Recovered	Ulcerative pyelitis and uræmia
67	0.08	0.06	Recovered	
97	0.135	0.25	Died	

cholesterol fell to 0.09 per cent within two days. Post-mortem examination showed a marked degree of ulcerative pyelitis, although this could not be regarded as the sole cause of death.

In addition to these cases, three of the patients with prostatic enlargement suffered from acute retention of urine on admission to hospital. In each case the initial finding was 0.12 to 0.13 per cent. Operative relief of the obstruction was followed within a few days by a rise in the plasma-cholesterol of between 0.03 and 0.07 per cent. This point is illustrated in Fig. 207, to which previous reference has been made.

CONCLUSIONS.

It will be seen, therefore, that the results obtained in this short series of cases do not bear out the claims made by MacAdam and Shiskin as to the importance of the plasma-cholesterol from a prognostic point of view.

The chief difficulties in the interpretation of the results appear to be the very wide range of variation which may be met with in normal people, and

the consequent impossibility of gauging the level in health for any given case. Of the other factors, acute retention of urine appears to have considerably more effect in lowering the plasma-cholesterol than is at present realized, so that deductions should not be drawn from results obtained only previous to the relief of the obstruction.

The impression gained from doing the actual investigations was that clinical observation, combined with blood-urea and urea-concentration observations, yielded in most cases a reasonably accurate forecast of future events. On several occasions a low plasma-cholesterol suggested a bad prognosis which did not materialize, whereas other patients with a normal cholesterol figure but clinically bad operative risks were either refused operation on clinical grounds alone or died shortly thereafter as a result of some complicating infection.

It would seem that so many factors are concerned in regulating the cholesterol content of the blood-plasma that it is impossible, in the present rudimentary state of our knowledge, to achieve an accurate summing up, with consequent enlightenment, from the consideration of a single figure obtained from any individual case. What is really needed is, first, a more intensive study of the normal figures obtained in healthy persons; secondly, more full investigation of the factors causing changes in the plasma-cholesterol; and lastly, a closer study of cholesterol findings and of cholesterol curves in different pathological states. It would be unjust to refuse operative assistance to any patient suffering from the effects of urinary obstruction merely because on one occasion previous to the proposed operation a low plasma-cholesterol figure had been reported. In my experience such results are extremely apt to be equivocal, and their interpretation is exceedingly open to fallacy.

The investigations described were performed in the laboratories of the Medical Professorial Unit during my tenure of the Baly Research Scholarship. I am indebted to the Surgeons to the Hospital for permission to investigate their cases, and to Professor F. R. Fraser for much helpful advice and criticism.

REFERENCES.

- ¹ MACADAM, W., and SHISKIN, C., *Brit. Jour. Surg.*, 1925, xii, 435.
- ² MAXWELL, J., *Quart. Jour. Med.*, 1928, lxxii, 297.
- ³ MACADAM, W., and SHISKIN, C., *Ibid.*, 1923, lxiii, 193.
- ⁴ GARDNER, J. A., and GAINSBOROUGH, H., *Biochem. Jour.*, 1927, xxi, 130.
- ⁵ LANDAU, *Deut. med. Woch.*, 1913, xxxix, 546.
- ⁶ CAMPBELL, J. M. H., *Quart. Jour. Med.*, 1925, lxxii, 393.
- ⁷ LUDEN, *Jour. of Lab. and Clin. Med.*, 1918, iv, 849.
- ⁸ BRUNTON, C. E., *Quart. Jour. Med.*, 1927, lxxix, 321.

THE ANATOMY OF CERVICAL RIB: WITH A REPORT OF A CASE.

By E. W. RICHES,

SURGICAL REGISTRAR TO THE MIDDLESEX HOSPITAL, LONDON.

It is generally accepted that there are two main types of cervical rib: the complete, where the rib is entirely bony and articulates with the first dorsal rib or with the sternum; and the incomplete, where the additional rib is short and is prolonged forwards by means of a fibrous cord. Wood-Jones¹



FIG. 212.—Skiagram showing bilateral cervical rib.

states: "Beyond the tip of the bony cervical rib . . . a ligamentous structure generally connects the rudimentary rib either to the first rib or to the sternum". Sargent² has carefully observed the non-ossified portion of the rib in twenty-two cases, and comes to the conclusion that it is a dense fibrous cord embedded in muscle, attached above to the rudimentary bone, whilst below it is most commonly attached to the first dorsal rib behind the sulcus subclaviæ. He has also observed that the band is tightened by inspiration and also by traction upon the arm, and has demonstrated this fact several times by completely detaching the cervical rib from its vertebræ and noting that it is pulled away from its former level when an inspiration occurs.

The case now described appears worthy of record in that the 'fibrous cord' consisted entirely of a muscle with a tendinous origin and a fleshy insertion.

HISTORY.—The patient was a girl of 21, a seamstress by occupation. In May, 1926, she was admitted to the Middlesex Hospital under the care of Dr. Voeleker, suffering from moderately severe Graves' disease. After two months of medical treatment, including radium, she was transferred to a surgical ward. In August Mr. A. E. Webb-Johnson ligatured the superior thyroid artery on each side, and one month later he removed the right lobe of the thyroid gland. These operations were highly successful, and in September she returned home much improved.

About a month later she noticed that she was losing the use of her right



FIG. 213.—Microscopic section from the right side showing bundles of striated muscle.

thumb, and then her right hand became weaker. She also had tinglings along the ulnar border of her right hand and forearm. On attending the outpatient department in December she was found to have wasting of the muscles of the right thenar eminence, and of the interossei, and about 50 per cent loss of power of the right hand. There was slight hypo-aesthesia on the ulnar border of the wrist and hand. These signs were thought to indicate the presence of a cervical rib, and an X-ray examination showed that there was a short cervical rib on the right side, and also a similar condition on the left side (*Fig. 212*). In March, 1927, she was re-admitted to hospital under the care of Mr. Webb-Johnson.

Despite the considerable wasting and loss of power all the muscles of the right hand were found to respond to faradism.

OPERATION.—On March 11, 1927, Mr. Webb-Johnson removed the cervical rib on the right side. An incision was made just above and parallel to the clavicle, reaching from the sternomastoid to the trapezius; when the brachial plexus was retracted downwards 'a firm fibrous band' was found running downwards from the tip of the rib; this was divided, and the rib cut off close to its neck with bone forceps.

The fibrous tissue was sectioned in the Bland-Sutton Institute of Pathology as a routine measure, and on microscopic examination was found to consist of bundles of striated muscle fibres (*Fig. 213*).

This operation was well borne by the patient; the wound healed by first intention, and considerable recovery of power in the hand was soon manifest.

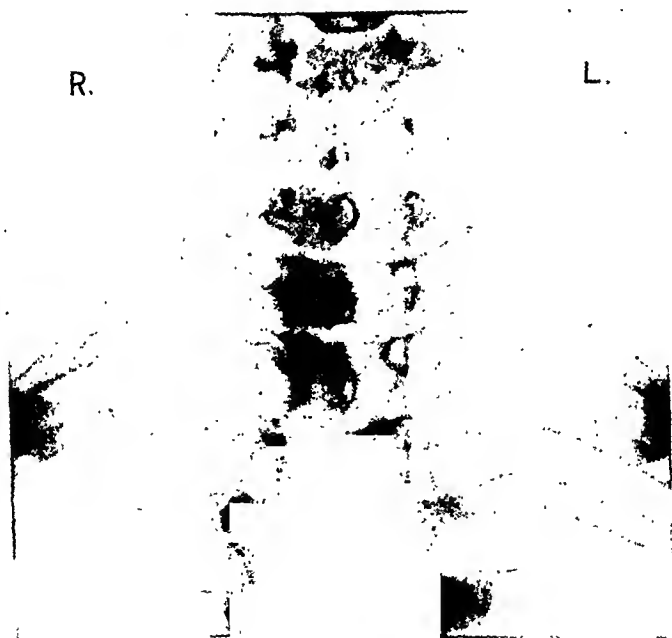


FIG. 214.—To show the condition of the patient after removal of both cervical ribs.

It became evident that pressure symptoms were increasing on the left side; loss of power was progressive, there was some wasting, and paræsthesia along the ulnar border. It was therefore decided to remove the left rib also, and on April 27 this was done by Mr. Webb-Johnson.

A similar incision was made above the left clavicle; the brachial plexus was retracted downwards and the cervical rib exposed. It appeared to point downwards, and from its tip sprang a strong fibrous band; this was traced down and found to end in a muscle belly which was inserted into the first dorsal rib. The muscle was divided as low as possible, and the tip of the cervical rib removed with bone forceps. *Fig. 214* shows the condition after operation.

The patient was discharged from hospital on May 23, but continued to

have massage. The power of the hands returned, and she resumed work in October. When seen in April, 1928, about a year after the operations, she had very good power in both hands, but the wasting remained unchanged. Her only complaint was of susceptibility to cold, and of some diminution of power in her hands when they were cold.

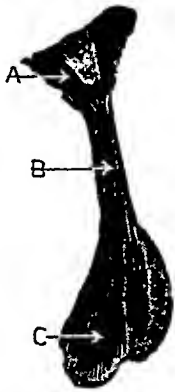


FIG. 215.—Specimen showing: A, Tip of cervical rib; B, Tendinous band; C, Muscular belly.

DESCRIPTION OF THE SPECIMEN.—The specimen removed (*Fig. 215*) shows the extremity of the cervical rib, which has been cut in section. Springing from the tip is a tendinous band which runs down for about an inch and then gradually broadens into a fleshy muscular belly. About three-quarters of an inch of this is shown, and its width at the lower end is about half an inch. The tendinous fibres are continued on the anterior surface of the muscle, most of the fleshy fibres arising posteriorly. There are two other small muscular slips connected with the posterior surface of the cervical rib.

DISCUSSION.

It would appear that this muscle is of the nature of a scalene, or rather an intercostal, the representative of a scalene in the thorax. It arises from a true costal element, and so cannot be regarded as the scalenus pleuralis (Sibson), which arises when present from the transverse process of the 7th cervical vertebra, and not from the anterior tubercle or costal element.

Professor Yeates, who has seen the specimen, is of the opinion that it is to be looked on as an external intercostal muscle.

It would seem probable that the nerve-pressure symptoms were in part due to the respiratory contraction of this muscle rather than to simple pressure of a fibrous cord.

I am indebted to Mr. Webb-Johnson for permission to publish the case, and to Professor McIntosh, Director of the Bland-Sutton Institute of Pathology, for permission to use the specimen and microphotograph.

REFERENCES.

- ¹ WOOD-JONES, *Proc. Roy. Soc. Med. (Clin. Sect.)*, 1913, Feb. 14, 95.
- ² SARGENT, Sir PERCY, *Ibid.*, 122.

THE TECHNIQUE AND RESULTS OF PARTIAL GASTRECTOMY FOR CHRONIC GASTRIC ULCER.*

By JOHN MORLEY,

HONORARY ASSISTANT SURGEON TO THE MANCHESTER ROYAL INFIRMARY.

WITH A NOTE ON GASTRIC ANALYSIS FOLLOWING PARTIAL GASTRECTOMY.

By W. MORRELL ROBERTS,

DIRECTOR OF THE CLINICAL RESEARCH LABORATORY OF THE MANCHESTER ROYAL INFIRMARY.

MANY discussions on partial gastrectomy have been held in the past few years, and it is a fair criticism of most of them to say that the interest of surgeons has centred chiefly on questions of operative technique and immediate operative mortality, while too little attention has been paid to the more remote effects of gastrectomy upon the patients' general health. The impetus towards the vogue of partial gastrectomy for simple ulcer of the stomach was undoubtedly provided by extravagant statements as to the frequency with which cancer supervened on simple gastric ulcer. Many surgeons conceived it their duty to perform extensive resection of the stomach for chronic ulcers that they believed to be precancerous lesions. Others of us, while unwilling to accept pathological views of the relation of ulcer and cancer that seemed to be in direct conflict with our clinical experience,¹ yet were so impressed by the smooth and easy convalescence from a Pólya gastrectomy, the immediate relief from pain, and the rapid gain in weight that often follows, that we were inclined to regard it as the operation of choice, and the stomach as an organ that could be sacrificed without serious inconvenience to our patients. For one must insist at the outset that a Pólya gastrectomy does as a rule abolish gastric digestion completely. The food, with such little gastric juice as continues to be secreted, drops straight through into the jejunum without even a transient hold-up in the stomach, and all that the gastric tube withdraws is a little alkaline and bilious jejunal content.

My purpose in the remarks that follow is, in the first place, to plead for a more critical attitude towards the Pólya operation as applied to simple ulcer. I have for some years been impressed by the fact that a certain proportion of patients after a Pólya gastrectomy, though relieved of their old pain and vomiting, and perhaps expressing themselves as satisfied with the result, yet showed a marked tendency to anæmia. This tendency was most noticeable in those who were anæmic at the time of operation as a result of repeated pre-operative hæmatemeses. Such cases seemed to be under some

* The opening paper of a discussion on partial gastrectomy by the Manchester Surgical Society, on March 20, 1928.

handicap that prevented them from regenerating blood as they ought, and in spite of medical treatment they continued to present a lemon-yellow colour and some shortness of breath on exertion.

Hartman² in 1921 reported a case in which a total gastrectomy by W. J. Mayo for cancer was followed twelve months later by an anæmia in which the blood picture was typical of pernicious anæmia. Hartman also referred to a case reported by Moynihan in 1911, where a man died of anæmia without recurrence of growth, three years and eight months after a total gastrectomy for cancer. He suggests that, had a blood examination been made, this anæmia might have been found to be pernicious. Hurst³ in 1924 reported a private communication to him by J. R. Bell of three other cases of Addison's anæmia following total gastrectomy for cancer in Vienna. Perhaps of equal significance is a case reported by Conybeare⁴ in which pernicious anæmia followed a gastro-enterostomy for gastric ulcer, and Hurst (*loc. cit.*) mentions a similar instance in the practice of Sir William Willcox. These cases, though undoubtedly rare, are yet numerous enough to suggest a very definite significance. It has long been recognized that achlorhydria is a constant feature of pernicious anæmia. Hurst believes that achlorhydria is not only a concomitant, but an essential predisposing cause, of pernicious anæmia. He quotes Faber, who found three cases of complete achylia with normal blood. Ten, seven, and three years later they developed typical Addison's anæmia. Numerous similar instances have since been reported, and it is well recognized that pernicious anæmia is sometimes preceded by an anæmia of the 'secondary' type. It would appear that the achlorhydria which predisposes to pernicious anæmia may be caused by any condition in which such acid as is secreted is at once completely neutralized. In the Pólya gastrectomy, particularly when it is performed for cancer or for gastric ulcer, the achlorhydria is as a rule complete.

The rôle of the gastric juice in warding off pernicious anæmia or, conversely, the manner in which achlorhydria predisposes to that disease, has engaged the attention of various investigators. Knott⁵ found that in over 90 per cent of cases showing a normal quantity of free hydrochloric acid in the gastric juice, the duodenal contents were sterile. Where achlorhydria was present, on the other hand, the duodenal contents were always teeming with organisms which had a tendency to be faecal in type. Knott believes that under normal conditions waves of acidity keep down these faecal organisms, preventing their growth in the upper reaches of the small intestine. His work is in full accord with the conception that the hæmolysis of pernicious anæmia is due to bacterial toxins produced in the intestinal tract.

One or two points in gastric physiology must be referred to by way of introduction to the subject of gastrectomy. The teaching of Edkins, that the mucosa of the pyloric antrum alone forms the specific hormone or gastrin which stimulates the flow of gastric juice from the upper portion of the stomach, has not been confirmed by later investigators. Tomaschewsky,⁶ for example, found that gastrin is distributed uniformly throughout the gastric mucosa. Steinberg, Brougher, and Vidgoff⁷ found that when the fundus was separated from the antrum in dogs and various substances were introduced into the fundus, the fundus glands gave a highly acid gastric juice.

It is most important in our discussion to visualize the exact distribution of the acid-secreting glands in the human stomach. The distribution of the oxyntic cells in the human stomach has been worked out by Miyagawa.⁸ He found that the pyloric glands, containing very few oxyntic cells, extend three-tenths of the distance from the pylorus to the cardia, and are of the same extent on the anterior and posterior walls. After a transitional area 1 cm. wide, there comes the large area of the unfortunately named 'fundus glands', rich in oxyntic cells (*Fig. 216*). The important point is that the oxyntic cells are massed chiefly in the body and central region of the stomach, and are scanty both in the fundus proper and in the pyloric regions.

We have, however, to consider a problem that is both wider and more complicated than the effect of gastrectomy on the gastric secretion. In doing these operations we have for some years been performing physiological experiments of a very important kind upon our patients, and it is only right that we should be at some pains to assess their results. In other words, we should try to estimate as precisely as possible the effects on our patients of the operation of partial gastrectomy. Before approaching that part of the paper, however, I must say something about the technique.

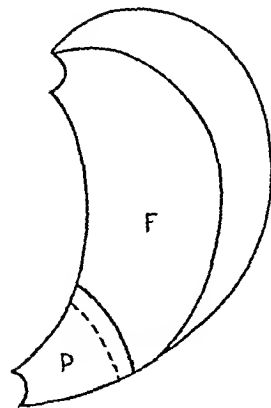


FIG. 216.—Distribution of glands in human stomach (Miyagawa). P, Pyloric; F, Fundal (oxyntic).

THE TECHNIQUE OF PARTIAL GASTRECTOMY.

On the technique of the Pólya gastrectomy I do not intend to dwell. It has become thoroughly standardized, and most surgeons will agree that the precolic modification is the simpler and more satisfactory form of this operation. On various grounds, some of which I have already mentioned, I believe that where simple ulcer of the stomach is concerned we have available a sounder and more physiological form of partial gastrectomy in the modification of the Billroth I operation introduced some years ago by Schoemaker, of the Hague.⁹ My earlier Schoemaker operations were performed with the crushing clamp introduced by Schoemaker, and I was soon impressed by the difficulty that I experienced, in all but very low ulcers, in applying this clamp sufficiently high up. Schoemaker's clamp is a somewhat clumsy instrument, and its mode of application across the stomach from the greater curvature is by no means easy. Souttar's modification¹⁰ is undoubtedly an improvement on Schoemaker's clamp, but shares the disadvantage that it must be applied from the greater curvature. It seemed to me that a crushing clamp was needed which could be applied from the upper border or lesser curvature of the stomach, and I invoked the mechanical ingenuity of Dr. K. B. Pinson, Anaesthetist to the Manchester Royal Infirmary, to design such an instrument¹¹ (*Fig. 217*).

The operation is begun by dividing the lesser omentum above the pylorus, and the gastrocolic ligament from below the first part of the duodenum to a

point as far to the left along the greater curvature of the stomach as is expedient. Two small Schoemaker's colectomy forceps are then applied side by side to the duodenum immediately beyond the pylorus. The duodenum is divided by cutting with a scalpel between these, and both forceps are protected by swabs. The stomach is now turned over to the left, and the lesser omentum with the coronary or left gastric vessels is divided and the vessels are tied. Another Schoemaker's colectomy forceps is applied to the stomach at right angles to the greater curvature at the point selected for end-to-end union with the duodenum. The large crushing clamp is now applied from the upper margin of the stomach so that it grips the stomach well above the ulcer, while the tip of its blades make contact with the tip of the small Schoemaker forceps (*Fig. 217*). An ordinary curved gastro-enterostomy clamp is applied distally to these clamps to prevent escape of contents, and the stomach is divided by cutting with a scalpel close to the sides of the crushing clamp and of the small colon forceps.

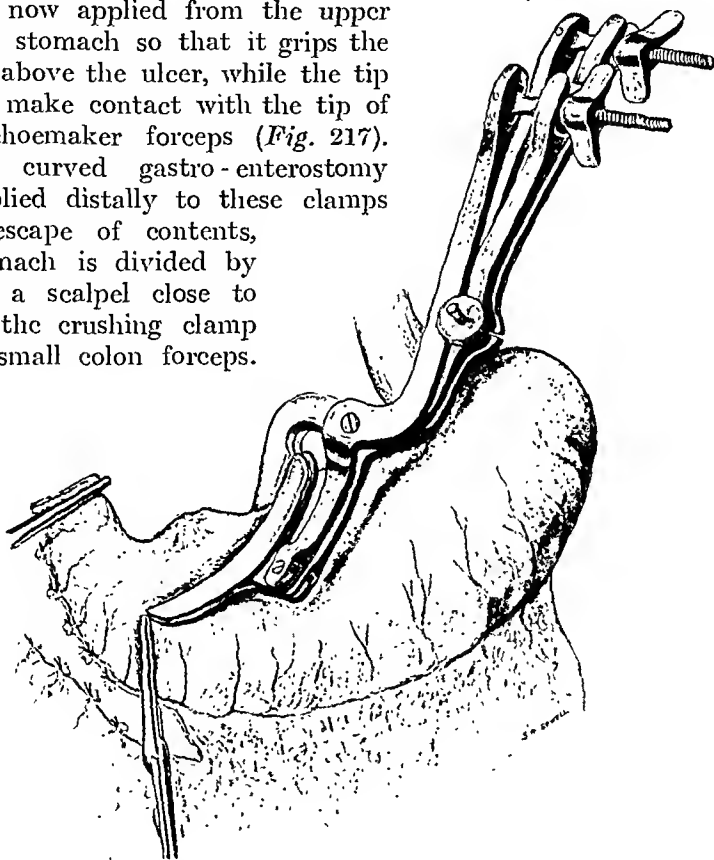


FIG. 217.—Method of applying the author's crushing clamp. The duodenum has been divided between clamps, and the crushing clamp and a Schoemaker's colectomy forceps are applied to the stomach.

The inner blade of the crushing clamp is now detached. A crushed frill of stomach wall is left projecting from the grip of the outer blade, and this is sutured with continuous fine catgut on a curved intestinal needle (*Fig. 218*). The outer blade is next removed, and a second invaginating catgut suture completes the closure of what is to be the new lower portion of the lesser curvature (*Figs. 219, 220*). The two colectomy forceps, one on the stomach and one on the duodenum, are now easily approximated, and end-to-end union

with catgut is completed. I first insert a continuous catgut stitch for the posterior portion of the seromuscular layer (*Fig. 221*), beginning at the upper

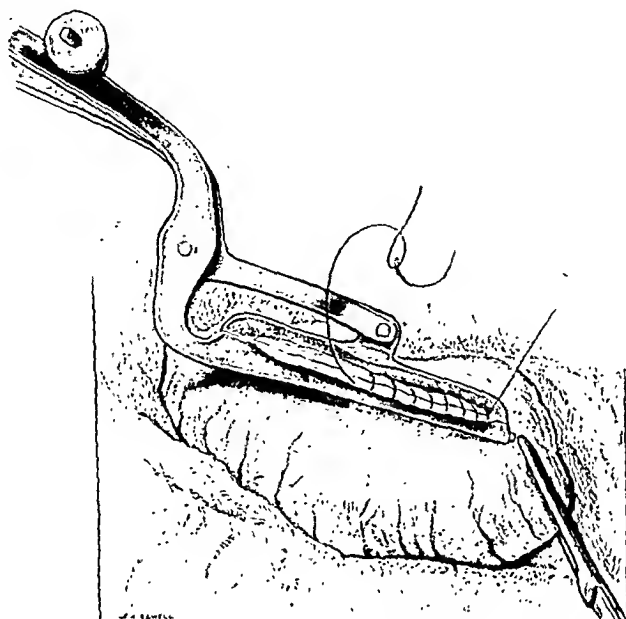


FIG. 218.—One blade of the crushing clamp has been removed, and the continuous haemostatic suture along the new lesser curvature is being inserted.

margin with a curved needle, and entering the needle parallel to the blades of the forceps. The suture is knotted at the lower margin and laid aside. Two

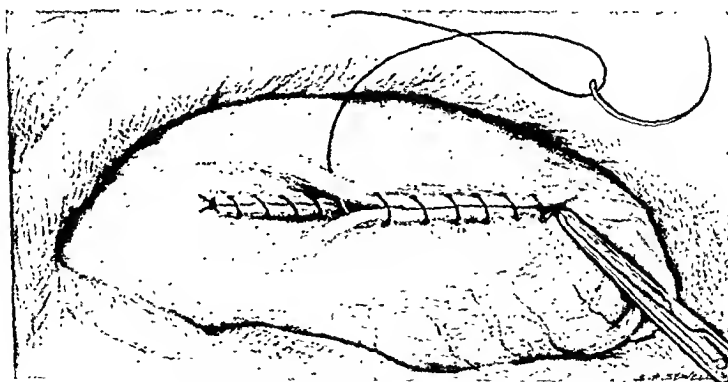


FIG. 219.—Application of the invaginating seromuscular suture along the new lesser curvature.

curved gastro-enterostomy clamps are applied, one to the new pre-pyloric portion of the stomach and one to the duodenum, and the two Schoemaker colectomy forceps are removed. A circular continuous stitch of catgut is now

inserted for the through-and-through layer (*Figs. 222, 223*). The seromuscular stitch is then picked up and its anterior half is completed (*Fig. 224*).

One or two points in this operation require emphasis. The crushing clamp must not be applied too obliquely, or a long and narrow tubular prepyloric portion of the newly constituted stomach will result, and this is apt to

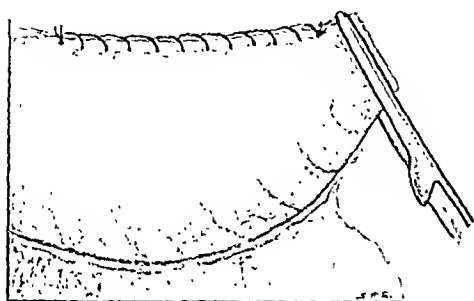


FIG. 220.—The new lesser curvature on completion of the suture: viewed from behind.

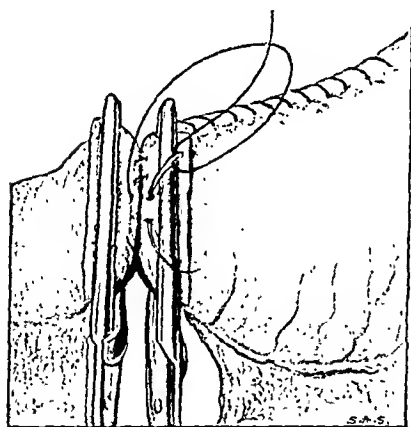


FIG. 221.—End-to-end union of stomach and duodenum: commencement of posterior row of seromuscular suture.

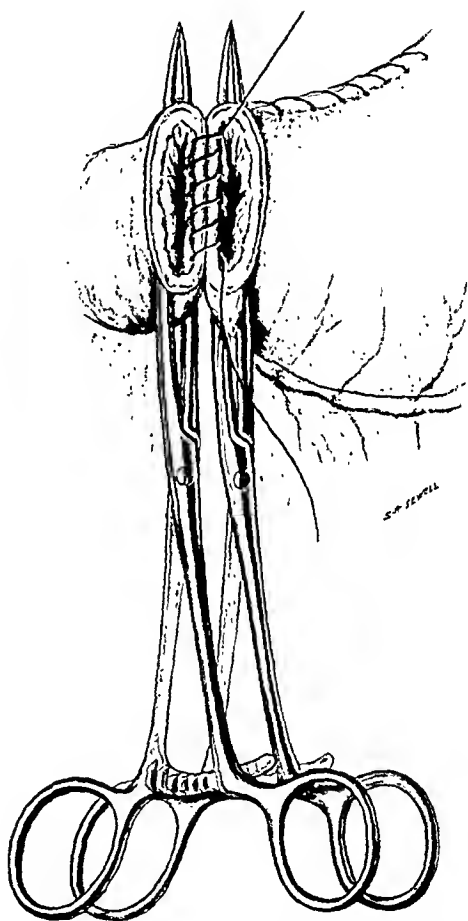


FIG. 222.—End-to-end union of stomach and duodenum: commencement of continuous haemostatic suture. For the sake of clearness the posterior half of the seromuscular suture is not shown.

give rise to post-operative vomiting for the first few days. There is no need to fall into this danger, for in cases where the ulcer is high up, a point on the greater curvature well to the left can be selected for the line of union with the duodenum. Owing to the length of the greater curvature and the ease with which it rotates towards the duodenum, there is never the least difficulty from tension at the site of end-to-end union. The gastroduodenal anastomosis

must be carried out with neat and accurate sutures, or some stenosis of the new 'pylorus' may result. It should always be possible for the operator's finger and thumb to meet with ease through the new 'pylorus', when testing its size by invaginating the walls at the conclusion of the operation. It need hardly be added that the greatest care must be exercised in suturing the classical 'dangerous angle' at the upper margin of the anastomosis.

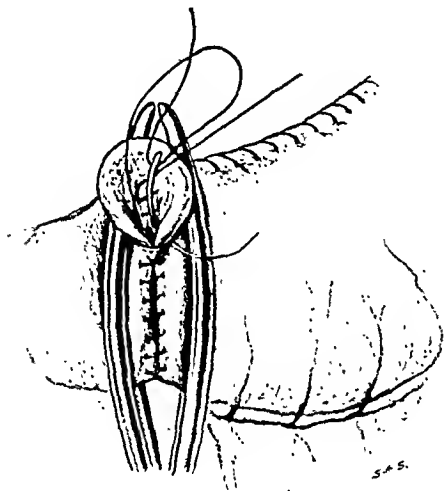


FIG. 223.—End-to-end union of stomach and duodenum: hæmostatic suture is shown nearing completion. For the sake of clearness the uncompleted seromuscular suture is not shown.

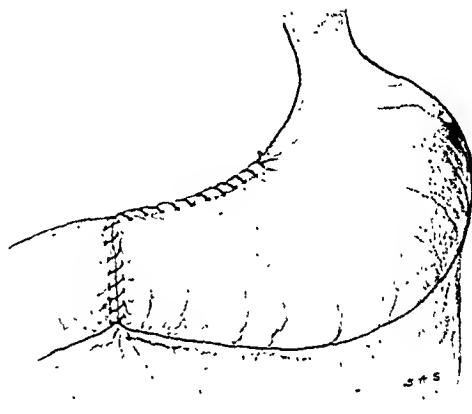


FIG. 224.—The operation completed, showing normal form of the stomach.

COMPARATIVE RESULTS OF OPERATION.

I will now compare the results of this Schoemaker operation with those of the more fashionable Pólya gastrectomy as far as my own experience is concerned. In doing so I must compare not only the immediate operative mortality but the post-operative condition of the patients.

Operative Mortality.—It is convenient to classify the Pólya and the Schoemaker operations into groups according to the conditions for which the operations were performed, viz., gastric ulcer and gastrojejunal ulcer. Gastrectomy for cancer is not considered in this communication, and I have not performed these operations for uncomplicated duodenal ulcer.

Pólya gastrectomy for gastric ulcer, 47 cases—3 died, mortality 6·4 per cent.

" " gastrojejunal ulcer, 5 cases—0 died.

Schoemaker " gastric ulcer, 68 cases—2 died, mortality 2·9 per cent.

Of the three fatal cases of Pólya gastrectomy, two early cases died from leakage at the invaginated end of the duodenum, and one from post-operative bronchopneumonia. Of the two fatal cases of Schoemaker gastrectomy, one died of acute pneumonia, and one, a man of very poor physique, died a few hours after operation of pulmonary œdema. In both cases post-mortem examinations were made, and there was no sign of leakage.

Remote Results of Gastrectomy for Gastric Ulcer.—If a case of Pólya gastrectomy is examined radiographically, the barium meal is seen to drop straight through into the jejunum with practically no retention in the stomach.



FIG. 225.—Stomach before operation, showing ulcer.



FIG. 226.—Same case as Fig. 225 after operation.

The stump of stomach that is left, unless the gastrectomy has been very limited in extent, acts merely as a prolongation of the œsophagus.

An X-ray examination, some considerable time after operation, has been

carried out in a large proportion of the Schoemaker gastrectomy patients, and the illustrations given are typical of the appearance found. In these cases we find from the X-ray examination that the food passes through the stomach very much more slowly than after the Pólya operation, though emptying is nearly always more rapid than in the case of the normal stomach. It is remarkable in many cases to observe how closely the picture resembles that of the normal stomach. A comparison of films taken before and after operation will illustrate this point (Figs. 225, 226). In some cases the appearance of a normal duodenal cap is reproduced (Fig. 227).

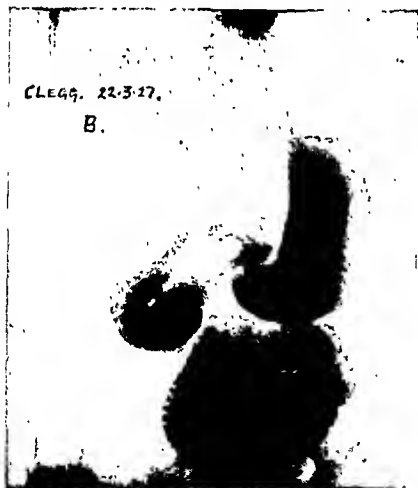


FIG. 227.—Stomach after operation, showing normal form of stomach and duodenal cap.

In attempting to assess the more remote effects of both operations, I was impressed by the need of something more precise and objective than a mere questionnaire about pain, appetite, and weight, and it seemed to me that an examination of the blood on the

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one hand, and a fractional test-meal on the other, might be carried out with advantage in as many patients as possible. This work has been very kindly done by Dr. W. M. Roberts, the Director of the Clinical Research Laboratory of the Manchester Royal Infirmary, and he will describe the results of his gastric analyses below.

Clinical Results.—The method of assessing the clinical results has been to send out a list of three definite questions to the patients. These were: (1) Has there been any return of pain or vomiting since the operation? (2) What is the state of your appetite? (3) How does your weight compare with before operation? In addition to this, as many patients as possible have been interviewed personally. Of those who attended, Dr. Roberts has examined the blood in both the Pólya and the Schoemaker group, and has performed fractional gastric analysis in the Schoemaker group only. It was found a waste of time to do this in the Pólya cases, as only a little bile could be withdrawn.

Table I.—COMPARISON OF PÓLYA AND SCHOEMAKER GASTRECTOMY RESULTS.

PÓLYA GASTRECTOMY FOR GASTRIC ULCER				SCHOEMAKER GASTRECTOMY FOR GASTRIC ULCER			
Cases	Blood examined	Anæmia	Results	Cases	Blood examined	Anæmia	Results
24	14	7	Good	37	18	2	Good
5	4	2	Fair	5	3	2	Fair
4	3	3	Poor	0	0	0	Poor
4*	—	—	Died since discharge	3	—	—	Died since discharge
7†	—	—	Untraced	8	—	—	Untraced
3	—	—	Died	2	—	—	Died
0	—	—	Too recent	13	—	—	Too recent
47	21	12		68	21	4	

* 1 clinically anæmic up to death two years after operation.

† 1 clinically very anæmic three years after operation.

An arbitrary standard of what constitutes 'anæmia' had to be adopted, and Dr. Roberts has taken a red corpuscle count of below 4,500,000, or a hæmoglobin percentage below 60 per cent, as the criterion of 'anæmia'. All the blood-counts have been performed by Dr. Roberts, and the same standard of anæmia has, of course, been applied to both the Pólya and the Schoemaker groups.

In summarizing the clinical results I have classified the cases into three classes: 'good', 'fair', and 'poor'. No cases operated on during the last six months have been taken into account. Most of the Pólya gastrectomies were performed at an earlier date than the Schoemaker operations, and all but three of the Pólya group were operated on more than four and less than eight years ago. I began to employ the Schoemaker operation in 1922, and have used it with increasing frequency since that year.

In the Pólya cases, of those traced we have: good 24, fair 5, and poor 4. Fourteen of the 'good' group had their blood examined, and of these

Table II.—DETAILS OF 21 POLYA GASTRECTOMY CASES WITH BL

PATIENT	DATE OF OPERATION	AGE	SEX	ANÆMIA AT OPERATION	PAIN OR VOMITING
(1) B. McB.	26/8/20	32	M	None	None
(2) M. G.	4/12/25	45	F	None	None
(3) J. E.	18/2/27	52	M	+ +	None
(4) M. W.	2/8/23	35	F	None	A little pain after food unless restricted diet
(5) F. C.	2/9/20	41	F	None	None
(6) N. B.	8/8/23	44	F	None	None. Occasional wind
(7) L. C.	16/4/24	63	F	+ +	Some pain and vomiting at interv
(8) M. H.	28/4/20	51	F	Not noted	None. Feels quite well
(9) J. S.	28/4/23	34	M	None	No
(10) J. S.	26/8/22	31	M	+	None
(11) S. E.	4/1/23	52	F	None	No
(12) A. C.	18/5/22	48	F	None	None
(13) J. W.	29/10/21	39	F	None	Slight pain after food. Occasio vomiting
(14) R. W.	1/6/20	34	M	Not noted	None
(15) J. C.	29/10/22	53	M	+	None
(16) M. B.	8/6/20	46	F	Not noted	No pain. Slight palpitation at food
(17) J. C.	2/5/20	23	M	Not noted	None
(18) M. S.	15/11/20	52	F	+	Much pain after food as a rule
(19) J. W.	17/6/20	43	M	+ +	Only a little wind at times
(20) M. M.	11/8/21	34	F	+ +	Occasional pain. Has only vomit occasionally
(21) E. P.	10/4/20	36	F	+	None whatever

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AMINATION: SHOWING CLINICAL STATE IN MARCH, 1928.

APPETITE	WEIGHT	BLOOD-COUNT			SUMMARY *
		R.B.C.	Hb. %	Colour Index	
Fairly good	Gained a few lb.	6,150,000	92	0.75	G
Good	Gained a few lb.	5,000,000	48	0.48	G A
Fairly good	Gained 3 stone	4,450,000, anisocytosis	52	0.58	G A
Moderately good	Gained a little	3,900,000	44	0.56	F A
Moderate	Gained a little	4,250,000	76	0.9	G A
Varied	Gained 2 stone	4,750,000	62	0.65	F
Improving	Increasing	3,650,000	56	0.77	F A
Normal	?	4,875,000	58	0.6	G A
Good	Increasing	4,825,000	92	0.95	G
Fair to good	Not weighed	5,350,000	66	0.62	G
Good	?	5,250,000, some aniso- and poikilocytosis	52	0.45	G A
Good	Same as before	5,600,000	83	0.74	G
Poor	About the same	4,262,500, anisocytosis and poikilocytosis	36	0.42	P A
Very good	Normal	5,250,000	80	0.74	G
Good	Same as before	4,930,000	70	0.71	G
Eats little but feels hungry	Gained	4,950,000	72	0.73	F
Fairly good	Gained 8 lb.	5,235,000	89	0.85	G
Very poor	Gained a little	4,120,000, anisocytosis and poikilocytosis	35	0.43	P A
Good. Takes small meals	?	4,200,000	75	0.89	G A
Not very good	Has lost 2 stone	3,600,000, anisocytosis	42	0.58	P A
Fair	About the same	4,300,000, anisocytosis and poikilocytosis	46	0.54	G A

* G = Good ; F = Fair ; P = Poor ; A = Anæmia.

7 showed anæmia. Only one of these was a recent case (13 months), and he was very anæmic at the time of operation. In the second group, of 5 'fair', 4 were examined and 2 were anæmic, and one more escaped that condition by a narrow margin. Out of 4 'poor', 3 were tested and all were markedly anæmic. Of the 4 cases that died since discharge from hospital, one, who committed suicide two years after operation, was clinically anæmic, and of the 7 untraced, one was known to be clinically very anæmic 3 years after operation. Details of the above cases are given in *Table II*. The statements as to anæmia at the time of operation are based on clinical data only, as unfortunately blood-counts were not made before operation.

Amongst the Schoemaker cases we have: good 37, fair 5, poor 0. Of the 37 'good', out of 18 examined, 2 were anæmic, but both were recent cases (12 months and 8 months respectively after operation) and both were profoundly anæmic at the time of operation as the result of recent hæmatemeses. In the 5 'fair' results, 3 were tested and 2 found to be anæmic. Of these 2 cases, one was operated on only 12 months ago and was anæmic before operation. The test-meal in this case shows complete post-operative achlorhydria. The other case is employed in the unhealthy occupation of billiard-marker, and is also suffering from bleeding piles and a tendency to alcoholism.

I conclude from these results that the Pólya operation carries with it a serious liability to post-operative anæmia, which we must attribute to the achlorhydria resulting from the operation. In all cases examined the anæmia has been of the secondary type, though in some anisocytosis and poikilocytosis were present. One would be by no means surprised, however, if some of these patients should in future develop the changes characteristic of pernicious anæmia.

The Schoemaker patients, on the other hand, show distinctly better clinical results and are markedly free from post-operative anæmia. With one possible exception the four cases of anæmia could be explained in a manner that exonerates the operation from any share in its causation. It may be claimed with some confidence that we have in this operation a procedure which is radical in that it removes the ulcer-bearing area and the pyloric sphincter and permanently lowers the acidity, while it is conservative in that it leaves a stomach which functions in a manner sufficiently akin to the normal organ. Not only is it physiologically sounder than the Pólya gastrectomy, but it has a somewhat lower mortality, gives better clinical results, and is on the whole both simpler and easier to perform.

Pólya Gastrectomy for Gastrojejunal Ulcer.—The small group of 5 Pólya gastrectomies for gastrojejunal ulcer are all somewhat recent, and the number is too small for any general conclusions. The clinical results are so far excellent in each case. The radical reduction of acidity which follows the Pólya gastrectomy (except in one case of this group, who still has hyperchlorhydria) is the strongest argument for it as the operation of choice in these very difficult cases.

I am greatly indebted to Dr. Margaret Greg, whose sketches made during the course of operations provided the basis of most of the drawings illustrating the operative technique, and also to Drs. Bromley, Gray, Paterson, and Twining for the help they have given in the X-ray examinations.

A NOTE ON THE RESULTS OF GASTRIC ANALYSIS FOLLOWING PARTIAL GASTRECTOMY.

Test-meals carried out on patients within six months after Schoemaker's gastrectomy in most cases showed achlorhydria, for in only 2 out of 14 cases examined did any secretion of acid occur. That the achlorhydria was due to the absence of secretion and not to neutralization of the acid after secretion was evident from the results of chloride estimations. The chlorides of the gastric contents include any hydrochloric acid secreted, whether still existing as such or neutralized and thus present as sodium chloride. In the cases of achlorhydria, the chloride concentration did not rise appreciably during the test, from which it may be inferred that no secretion took place.

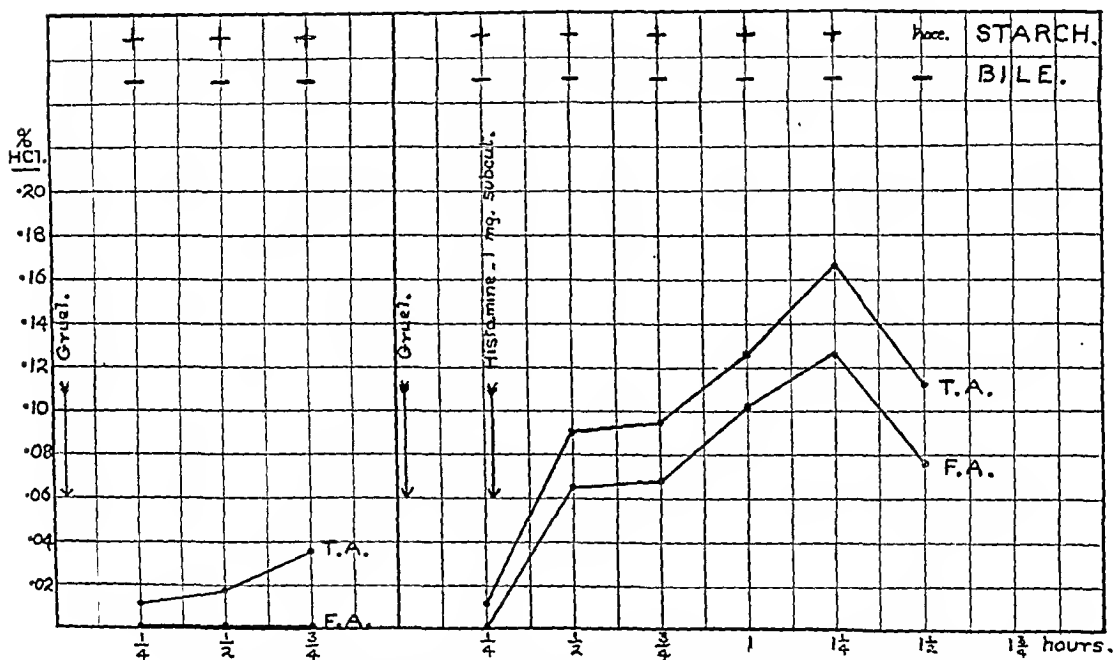


FIG. 228.—Chart illustrating gastric analysis after Schoemaker gastrectomy for gastric ulcer. Left-hand chart without histamine. Right-hand after 1 mgrm. histamine. T.A., Total acidity; F.A., Free acid.

The test was repeated on 6 of the achlorhydric cases, 1 mgrm. of histamine being injected subcutaneously as an additional stimulus to secretion. In these circumstances, one of the cases showed a definite rise in chloride concentration and three a secretion of acid, one of which reached normal figures. The chart (Fig. 228) illustrates the results obtained a few months after a Schoemaker gastrectomy in the case of a patient who, prior to operation, gave the hyposecretory type of curve, very common in ulcer of the lesser curvature. The first part of the test shows achlorhydria and rapid emptying, the lower curve (F.A.) representing the free acid and the upper curve (T.A.) the total acidity. The stomach having emptied of the gruel, a second portion was administered, followed, after the first sample had been withdrawn, by an injection of histamine, whereupon acid secretion occurred at once.

A result such as this indicates that the failure in secretion after the operation is due more probably to a disorganization of the reflex or hormonal relations between the different parts of the stomach than to the actual diminution in the area of the secreting surface. Further, cases examined at longer intervals after operation showed that the secretory capacity of the stomach had been largely or completely re-established, though there did not appear to be any clear relationship between the time that had elapsed since the operation and the degree of the secretory response.

Achlorhydria also follows Pólya gastrectomy, but test-meals give inconclusive evidence of whether or not this is to be attributed to neutralization of acid; for, as a rule, only small samples are obtained from the stomach, owing to the almost immediate emptying, and these contain a considerable proportion of bile. Since bile and intestinal secretions have a relatively high chloride concentration, the chloride percentage of a sample containing much bile cannot be regarded as a criterion of gastric secretion.

REFERENCES.

- ¹ MORLEY, J., *Lancet*, 1923, ii, 823.
- ² HARTMAN, J. T., *Amer. Jour. Med. Sci.*, 1921, clxii, 201.
- ³ HURST, A. F., *Medical Essays and Addresses*, London, 1924, 81.
- ⁴ CONYBEARE, J. J., *Guy's Hosp. Rep.*, 1922, lxii, 186.
- ⁵ KNOTT, F. A., *Ibid.*, 1927, lxxvii, 1.
- ⁶ TOMASCHIEWSKY, quoted by STEINBERG, BROUGHIER, and VIDGOFF (q.v.).
- ⁷ STEINBERG, M. E., BROUGHIER, J. C., and VIDGOFF, I. J., *Arch. of Surg.*, 1927, xv, 749.
- ⁸ MIYAGAWA, Y., *Jour. Anal.*, 1920-21, lv, 56.
- ⁹ SCHOEMAKER, J., *Surg. Gynecol. and Obst.*, 1921, Dec., 591.
- ¹⁰ SOUTTAR, H. S., *Brit. Med. Jour.*, 1927, i, 504.
- ¹¹ MORLEY, J., *Lancet*, 1925, ii, 554. (This clamp is now made by Messrs. Allen and Hanbury.)

THE SURGICAL TREATMENT OF GASTRIC ULCER, WITH SPECIAL REFERENCE TO THE MASSIVE ULCERS.*

By GARNETT WRIGHT,

HONORARY SURGEON TO THE SALFORD ROYAL HOSPITAL.

IN spite of the endless stream of literature on gastric ulcer, the subject remains a fascinating one. The obscure nature of the causes which produce and maintain these ulcers is responsible for a wide variation in the treatment, both medical and surgical. So long as these causes remain unknown, treatment must necessarily be empirical. I am concerned merely with surgical measures, which become necessary when medical treatment has failed permanently to heal the ulcer, when the lesion is of such a size that permanent healing is unlikely, when deformities of the stomach have been produced, and finally, when certain acute crises, such as hæmorrhage or perforation, have arisen. I do not propose to enter into the question of these acute complications, and will content myself by stating that when a patient has had a severe hæmorrhage from a chronic gastric ulcer, surgical intervention is imperative when the patient has sufficiently recovered for it to be undertaken safely. That intervention should include the removal of the ulcer, in order to prevent a recurrence of the hæmorrhage. Indirect methods, such as gastro-enterostomy, are not sufficient to ensure this.

Left untreated, or ineffectively treated, chronic gastric ulcer, after giving rise to much suffering and misery for many years, leads to death. This end may come as the result of one of the acute crises mentioned above—perforation into the peritoneal cavity, or hæmorrhage.

There is a tendency to minimize the risk to life from acute hæmatemesis, but there is no doubt that it is a real one, and that many sufferers from chronic ulcer succumb in this way. Bulmer, in a series of 249 cases of hæmatemesis from chronic ulcer, found a mortality of 11·6 per cent. It is interesting to notice that the mortality was higher in men than in women, and that several of the cases happened when gastro-enterostomy had previously been performed without healing the ulcer.

In old-standing ulceration deformities of the stomach are produced. The passage of the gastric contents into the duodenum becomes increasingly difficult, and the patient begins to suffer from under-nutrition, until he dies, worn out by pain and starvation. In some cases the end is hastened by pulmonary tuberculosis, which, if not actually caused by the starvation, is certainly made worse by it.

According to one school of thought, gastric ulcer commonly terminates

* A paper read before the Interstate Post-graduate Assembly, Kansas City, Missouri, October, 1927.

in carcinoma. This is a highly controversial question, and opinions are now veering round from the extreme views which were current some years ago. The Mayo Clinic in the United States, and Moynihan and Sherren in England, put forward the view that about 70 per cent of gastric ulcers become carcinomatous. This was based on the examination by McCarty of ulcers removed by operation. He found associated carcinoma in 68 per cent. Wilson and McCarty about the same time discovered evidence of previous chronic ulcer in 71 per cent of gastric carcinomata, while Smithies, working in the same clinic, found a clinical history of previous ulceration in 60 per cent of gastric cancers. From these very startling figures, supposing the facts are correct, the only logical deduction that can be drawn is that almost every chronic gastric ulcer must inevitably become malignant. This must be so, if so large a proportion as 68 per cent are carcinomatous at the time of operation, operated on, as they must be, in various stages of the disease.

The results of the treatment of gastric ulcer by gastro-enterostomy ought to be appalling, yet we find from the Mayo Clinic statistics (Balfour) only about 6 per cent of such cases subsequently developed carcinoma, and most of these died within two years of the operation—a fact which leads to the strong suspicion that they were cases of carcinoma at the time of the operation. Indeed, Balfour states that in many of the cases malignancy was suspected at operation. Bamberg followed up 1025 patients, of whom only 22 developed cancer. Again this happened mostly within two years, and the question of error in diagnosis arises.

Hartman, in reviewing the results of 187 cases of gastro-enterostomy for ulcer (72 pyloric stenosis, 115 ulcers without stenosis), found four deaths from cancer, one after 18 months, one after 3 years. Both of these he looks upon as having been cancer originally. One appeared after 7 years and one after 14 years. In the last case the cancer was not situated at the site of the original ulcer. This leaves only one case where cancer may have been grafted on a chronic ulcer.

This question was discussed at several meetings in Manchester some years ago, and different views were expressed. In 1920, at a meeting of the Manchester Pathological Society, Professor Dean suggested that in view of these differences of opinion a sub-committee be appointed to investigate the question. Material was provided by various Manchester surgeons and examined carefully by Dr. J. H. Dible, who was then Professor Dean's chief assistant in the Pathological Department of the Victoria University. The results of this investigation were published in the *BRITISH JOURNAL OF SURGERY* in 1925.¹ In 126 cases of chronic ulcer, malignant changes were discovered in none. In 33 cases of early cancer in only two did the weight of evidence point to malignant degeneration of a previously existing ulcer.

The clinical aspects of the cases were carefully analysed by Morley, whose findings agreed with the pathological evidence. The average pre-operation history in ulcer was ten years and four months; and in carcinoma two years and three months. The latter figure is unduly high owing to the presence of one case giving a history of twenty years, which in a small series (28) has an effect out of proportion to its importance. These facts, together with the different distribution of carcinoma and ulcer—carcinoma at the

pylorus, ulcer on the lesser curve—would make it appear that the transformation of a gastric ulcer into a carcinoma is a rare event, and this I believe to be the true state of affairs.

The surgical methods at our disposal in the treatment of chronic gastric ulcer may be classified into (1) *Indirect*; and (2) *Direct*, the former consisting of attempts to alter the physiology of the stomach, while the latter are directed to removal of the ulcer and in most cases also a modification of the physiology.

It is not my concern to review the numerous theories which have been propounded concerning the etiology of gastric ulcer. None of these is completely satisfactory and many are of little use in enabling us to find a completely satisfactory method of treatment. There are two factors, however, which by common consent have an important bearing on the persistence, if not the development, of ulcer. These are (1) The acid character of the gastric juice; and (2) Any interference with the emptying of the stomach, such as pyloric spasm. All methods of surgical treatment, with the exception of simple excision of the ulcer, are designed to modify these two factors, and therefore it is true to say that all methods are designed to modify the physiology of the stomach.

1. INDIRECT METHODS OF OPERATIVE TREATMENT.

The indirect methods at our disposal are gastro-enterostomy, some form of pyloroplasty, and jejunostomy.

Gastro-enterostomy has been the favourite in the past, and there is no doubt that it cures a large number of patients. The advocates of this operation claim 80 per cent of cures, while the opponents put the figure at round about 60 per cent. All surgeons must have seen cases where the ulcer has persisted in spite of gastro-enterostomy, and where acute crises have arisen. Hæmorrhage and even perforation may occur. I have had one patient in whom gastro-enterostomy had been done for duodenal ulcer. At the time of operation no gastric ulcer was noted, although the stomach was examined, yet eighteen months later the patient was admitted to hospital with a perforation of a small ulcer high up on the lesser curve, near the œsophagus. Gastro-enterostomy had been unable to prevent the formation of an ulcer in this patient.

The manner in which gastro-enterostomy acts in healing gastric ulcer is the subject of controversy. All are agreed that it acts mechanically by providing an efficient outlet from the stomach and obviating pyloric spasm. Paterson and many other surgeons believe that it has a physiological action also, allowing the regurgitation of bile and pancreatic juice into the stomach and diminishing the acidity of the gastric contents. He found that this acidity of the gastric contents was reduced below its pre-operative level after gastro-enterostomy. It is noteworthy, however, that this effect is most marked in cases of duodenal ulcer, and many observers have not found the lowering of acidity described by Paterson. It has been pointed out that a very large quantity of alkaline duodenal juice would be required to produce an appreciable lowering of the gastric acidity. Even the diversion of the whole of the duodenal contents into the stomach produced only a slight lowering of

acidity, and the same was true of the diversion of the bile into the stomach by means of cholecystgastrostomy in dogs (Wiedman and Enderlen).

Perman² examined thirteen cases of gastro-enterostomy and nine cases of gastric resection in which gastrectomy had been performed at the time of the operation. In none except the very extensive resections was there any diminution in acidity of the gastric juice in the early days after operation. It seems improbable, therefore, that gastro-enterostomy has any considerable effect on the gastric secretions, and this is the opinion expressed by Moynihan.

Sherren and others lay great stress on the position of the stoma in gastro-enterostomy for gastric ulcer, and are insistent that it should be on the proximal side of the ulcer. When we consider that most gastric ulcers are well away from the pylorus, it is evident that this means a stoma placed well towards the cardiac end of the stomach. The radiogram of a lesser-curve ulcer somewhat lower in level than the majority shows that in the upright position such a stoma would be well above the lowest part of the stomach. Hartman³ has shown that with a patent pylorus such a stoma hardly acts at all, most of the gastric contents passing through the pylorus. He showed quite clearly that when the opening was placed in the pyloric antrum the stomach emptied more rapidly, and that most of the gastric contents went through the gastro-enterostomy. It would appear therefore that, so far from being an advantage, the proximal position of the stoma is harmful and may almost entirely destroy the value of the operation. The opening should be placed so that the outlet is at the lowest point of the greater curvature, irrespective of the site of the ulcer.

Gastro-duodenostomy is an alternative operation to gastro-enterostomy. It has the advantage of delivering the gastric contents into the duodenum, but otherwise acts in the same manner as the former operation.

Pyloroplasty, either the old Heinecke-Miculicz operation or the slightly modified form of Shelton Horsley, or Finney's modification, may be employed, and would probably give similar results.

Jejunostomy enables one to feed the patient while resting the stomach, and would appear to be an ideal operation as an aid to medical treatment. It has proved somewhat disappointing, probably because the mere introduction of food into the jejunum excites gastric secretion, and the rest to the stomach is not so complete as might be expected.

All these indirect methods of treatment fail in a proportion of cases, and probably in a higher proportion than when the ulcer is removed. This is only to be expected, as the ulcer is bound to be replaced by a somewhat extensive scar, which must be liable to break down again under slight provocation. Most surgeons now believe that they get a higher percentage of cures after some form of resection.

2. DIRECT METHODS OF OPERATIVE TREATMENT.

RESECTION OF THE ULCER.

The simplest method is wedge resection of the ulcer. This was used at one time, but fell out of favour because of recurrence of ulceration. In Troell's statistics it is interesting to find that simple resection of the ulcer had

the highest mortality of all forms of operative treatment. A possible explanation lies in the nerve mechanism of the pylorus. Wedge excision necessitates the ligation of the nerves running along the lesser curvature of the stomach. McCrea and McSwiney have shown that the nerve-supply of the pylorus runs to the right in the lesser omentum, and then descends vertically to the pylorus. A ligation round the branches of the vagus on the lesser curve may lead to a reflex stimulating impulse which will then be carried along this pyloric nerve and produce a spasmodic closure of the pylorus and consequent increased intragastric tension, with possible leakage at the site of suture.

Most surgeons now combine this direct excision of the ulcer with one of the indirect operations—usually gastro-enterostomy, and more rarely pyloroplasty. In this way pyloric spasm is obviated and the reflux of duodenal contents is provided. Not only is all immediate danger of leakage from increased intragastric tension avoided, but the risk of recurrence of ulceration is minimized. This local excision and posterior gastro-enterostomy is the method advocated by Walton.

This operation, while easy in the case of ulcers situated on the distal part of the lesser curve, becomes increasingly difficult the nearer we approach the œsophagus, and in the case of very high ulcers it may be an extremely difficult procedure. Balfour has devised the cautery method for such cases. This consists in turning back a flap of the peritoneal coat of the stomach until the ulcer is exposed. The base of the ulcer is then burned out, the resulting gap in the stomach wall sutured, and the peritoneal flap replaced. My experience of this operation is limited to one case, where gastro-enterostomy had been done some years before by another surgeon for an ulcer on the lesser curve. The ulcer had not healed, and I treated it by Balfour's method. Eleven days later the patient had a very severe secondary hæmorrhage, obviously from the cauterized ulcer. Recovery followed, but the ulcer recurred. It seemed to me that the complication of secondary hæmorrhage was one which was not under my control, and therefore I have not used this operation since.

It is possible, by completely mobilizing the lesser curvature, to excise ulcers situated even near the œsophageal opening. In order to do this it is necessary to divide the coronary artery between two ligatures as it runs forwards in the left pancreatico-gastric fold from the posterior wall of the abdomen to reach the upper end of the lesser curvature of the stomach. The distal end of the vessel, together with the lesser omentum, is then stripped downwards along the lesser curvature until the ulcer is reached. In this way the lesser curve is freely mobilized and very high ulcers become accessible.

When the resulting gap from wedge excision of an ulcer is sutured, the lesser curvature is considerably shortened and the stomach becomes more globular in shape, with a tendency to kinking of the pylorus (*Figs. 229, 230*). This deformity can be obviated by removal of a corresponding portion of the greater curvature—in other words, by performing what is called a 'sleeve' resection, a complete circular segment of the stomach being removed. This form of local removal is, I think, the ideal one, although it suffers from the disadvantage that gastro-enterostomy becomes

difficult if not impossible. There is, however, an efficient substitute for gastro-enterostomy in pyloroplasty. In order to retain a normally shaped



FIGS. 229, 230.—Wedge resection, showing shortening of lesser curvature which results.

stomach a longer piece should be removed from the greater curvature than from the lesser (*Fig. 231*).

These local resections are specially suitable to ulcers of small size where we are anxious not to sacrifice a large area of healthy stomach. Sleeve resection is valuable in some cases of hour-glass contraction of the stomach, especially when the constriction is not too near the cardiac end.

In many cases of ulcer there is a longitudinal contraction of the lesser curve, so that the œsophageal and pyloric openings of the stomach become approximated. The stomach then assumes a globular shape and is known as the 'football' stomach. In such cases there may be extreme retention of stomach contents although the pylorus remains patent, owing to an acute kinking of the pylorus. Such a lesion is quite unsuitable for any kind of local excision, and should always be treated by subtotal gastrectomy.

For reasons given above, I believe that all forms of local excisions should be combined with gastro-enterostomy or pyloroplasty. Gastro-enterostomy is sometimes difficult and is not nearly so simply and quickly done as pyloroplasty, and for that reason I have usually employed pyloroplasty.

I have 21 cases of this operation, 9 men and 12 women. One of these died a fortnight after operation. The patient—a woman—had had a severe hæmatemesis a few weeks before operation and was very anæmic. She was recovering rather slowly, and my hospital resident gave her a blood transfusion

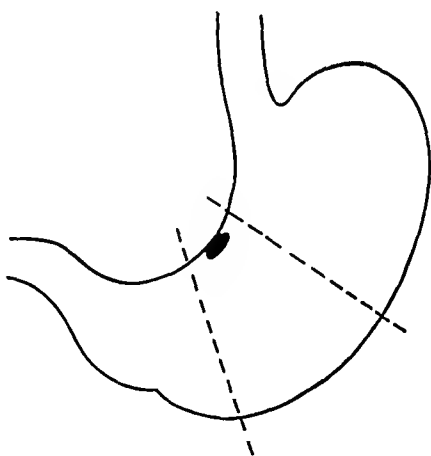


FIG. 231.—Lines for incision in ideal 'sleeve' resection.

in order to hasten her convalescence. Some error was made in the classification of the blood, as the patient quickly died from agglutination. Of the remaining 20 cases, 6 are quite recent. Of the 14 remaining, 1 has a definite recurrence of the ulcer, 1 has occasional slight pain, and the rest (12) are quite well. One patient had glycosuria before the operation and still has to have treatment for this; otherwise he is well.

Of local excision combined with gastro-enterostomy I have 5 cases; 4 are well. In one the ulcer recurred, and was cured by a Pólya partial gastrectomy.

GASTRECTOMY.

Resections of the stomach itself are advocated by some, notably Moynihan and Finsterer, as a routine treatment for gastric ulcer. The reasons given are that by this means gastric function is practically abolished and recurrence of ulceration prevented, the stomach being rendered incontinent and emptying itself almost at once into the intestine. Gastric stasis is entirely eliminated and the production of hydrochloric acid is much diminished. It is true that the portion of the stomach left behind is the acid-secreting part, but it is said that the hormones necessary to produce this secretion are manufactured in the pyloric end of the stomach. When this is removed these hormones are not produced, and therefore the secretion of acid is not called forth. In any case, a large portion of the gastric mucosa having been removed, much less gastric juice must be secreted, while the rapid emptying of the stomach prevents any accumulation of that which is formed. It would follow from this that the whole-hearted advocates of this method of treatment recommend a very large resection of the stomach, and this is so. Finsterer advocates a subtotal gastrectomy, and this is what is also figured in Moynihan's lectures on gastric ulcer.

The justification is held to be: (1) The fear of malignant changes in an ulcer; and (2) The freedom from recurrence of ulcer. I have already tried to show that the first reason is not a good one. I believe the latter one to be sound and reasonable.

The objections taken are that there is an unnecessary sacrifice of healthy stomach when the ulcer is of small or moderate size, and that the mortality must be very much higher than the more conservative operations. Hurst also draws attention to the risk of pernicious anaemia being set up by the absence of hydrochloric acid from the gastric juice.

The answer to the first objection is that if a large gastrectomy cures ulceration with certainty and leaves the patient in good health, it is justified, even if the ulcer be a small one. The risk of the operation has been exaggerated. Moynihan's series of 184 cases with 2 deaths shows this, and there can be no doubt that in ulcers of moderate size the operation is a very safe one. When very large ulcers, in patients broken down by months of pain and starvation, are operated on by this method, the risk is increased, but not so seriously as might be expected. This point will be dealt with more fully later. The risk of pernicious anaemia must be slight, as I have never seen it, though I have followed up a fair number of cases, many of whom have had high resections.

The methods of gastric resection at our disposal may be classified under the headings of Billroth No. I and Billroth No. II.

The Billroth I methods entail an end-to-end union of the cut end of the stomach with the duodenum. At one time this could only be attempted with very limited resections, but the Schoemaker technique has extended the sphere of usefulness of this operation very considerably. The advantages claimed for it are that it is more physiological than the other methods, the gastric contents being delivered into the duodenum. While this is true, it is arguable that since our efforts are directed to altering the physiology, the methods which do this most efficiently are to be preferred. It is also claimed that more stomach can be saved by this method than by the Pólya operation. The saving, however, is less than one would at first sight expect. It is essential that the incision from the lesser curvature should not be too oblique; otherwise a very long and narrow tube is left to be united to the duodenum. In actual practice the section of the stomach is much less oblique than diagrams would lead one to suppose, and the amount of stomach remaining is not very much greater than after a resection by the Pólya method. I have done this operation on two occasions only, and found the application of the special clamps a matter of such extreme difficulty that I put them on one side and used ordinary stomach clamps. One patient was operated on six months ago, and is now in excellent health. The other is a recent case, and is well so far. I personally am inclined to use the operation as an alternative to resection where the ulcers are of small and moderate size, and when they are situated not too high on the lesser curve.

The Billroth II methods are: (1) The original one of closure of the duodenum and the cut end of the stomach, and the restoration of continuity by means of gastro-enterostomy; and (2) Closure of the duodenal stump and implantation of the cut end of the stomach into the side of the jejunum by the method of Pólya, or preferably by Mayo's modification, where the jejunum is brought in front of the colon and made to run from the greater to the lesser curvature of the stomach. This last is the operation of choice, and is to be employed in all ulcers of large size, in all cases of recurrent ulcer, and in gastrojejunal ulcer. It is applicable to very large ulcers where the lesser curvature is shortened and there is very little healthy stomach above the ulcer.

This operation gives excellent functional results, and in moderate-sized ulcers is attended with very little risk. In cases of extremely large and complicated ulcers of the stomach most surgeons adopt the method of Moynihan, who performs a gastro-enterostomy en-Y, combined with jejunostomy. By this means he produces healing of the ulcer, though a very long time elapses before this is complete, sometimes more than twelve months, during which time the jejunostomy has to be kept open. Healing in such a case must result in a large area of scar tissue in the stomach, which must be liable to break down again.

For some years now I have been in the habit of subjecting ulcer patients to partial gastrectomy, with the result that, at the expense of a slightly increased risk, they are rapidly and permanently cured. The increase in risk is not so great as might be expected, and it is wonderful to find what a severe

operation a patient can survive although broken down and emaciated by long pain and starvation. These people have very much more reserve to draw on than have those who are the victims of carcinoma, and the mortality statistics of partial gastrectomy in the latter condition are of no value in assessing the risks of operation in ulcer cases.

The value of this operation in massive ulcers was first borne in on me by the case of a man 38 years of age. For five years he had had pain; for the last few months before operation this had been associated with vomiting of the cumulative type, and for some time very little of the stomach contents could have been passing onwards. He was so emaciated by starvation that

he resembled the pictures of victims of the Russian famine which were then appearing in the illustrated papers, and he was nicknamed the 'Russian famine' in the hospital. The accompanying photograph was taken about a fortnight after operation, and shows well the extreme degree of emaciation to which he was reduced (*Fig. 232*). I was asked to see him by a colleague, whose diagnosis was carcinoma of the pylorus, and this was supported by the X-ray findings. The radiogram shows great dilatation of the stomach and apparently a filling defect at the pyloric end.

From the fact that this patient had had symptoms for five years I doubted the diagnosis of carcinoma, believing, as I have said, that carcinoma rarely supervenes on ulcer. He also had a definite tender swelling under the left rectus muscle which made me think he had an ulcer of the lesser curvature adherent to the abdominal wall. I decided to operate on him, and did



FIG. 232.—Photograph showing the extreme degree of emaciation associated with a massive gastric ulcer.

so on March 26, 1922. I found the stomach adherent to the back of the left rectus abdominis. After separating the adhesions I found a chronic perforation of the stomach into the abdominal wall at this point. With considerable difficulty the stomach was then freed from the liver, the pancreas, and the mesocolon. *Figs. 233 and 234* are from drawings taken of the stomach some years later, after it had been hardened in formalin. They show that there has been an enormous saddle-shaped ulcer of the lesser curvature. The anterior portion of the ulcer has perforated the stomach wall, the greater part of the perforation being closed in by a false fibrous wall, except where it has penetrated the abdominal wall. The posterior part of the ulcer has also

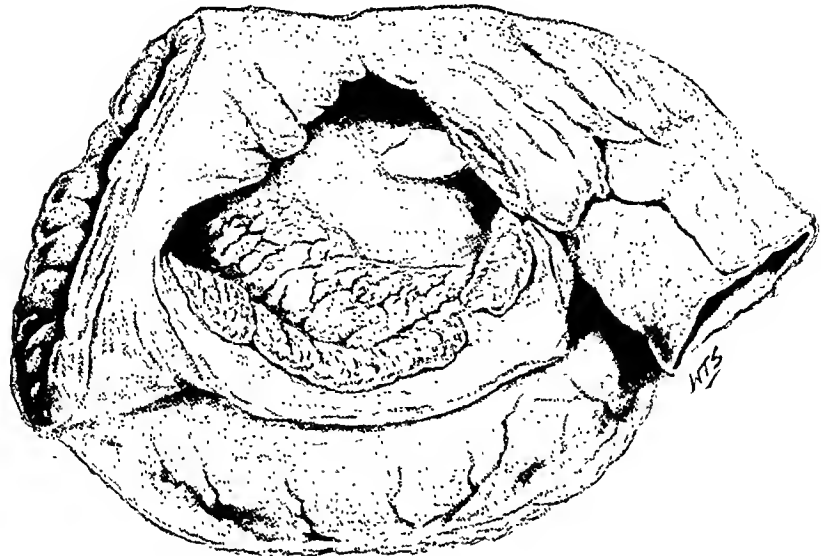


FIG. 233.—The same case as *Fig. 232*, showing posterior view of stomach.

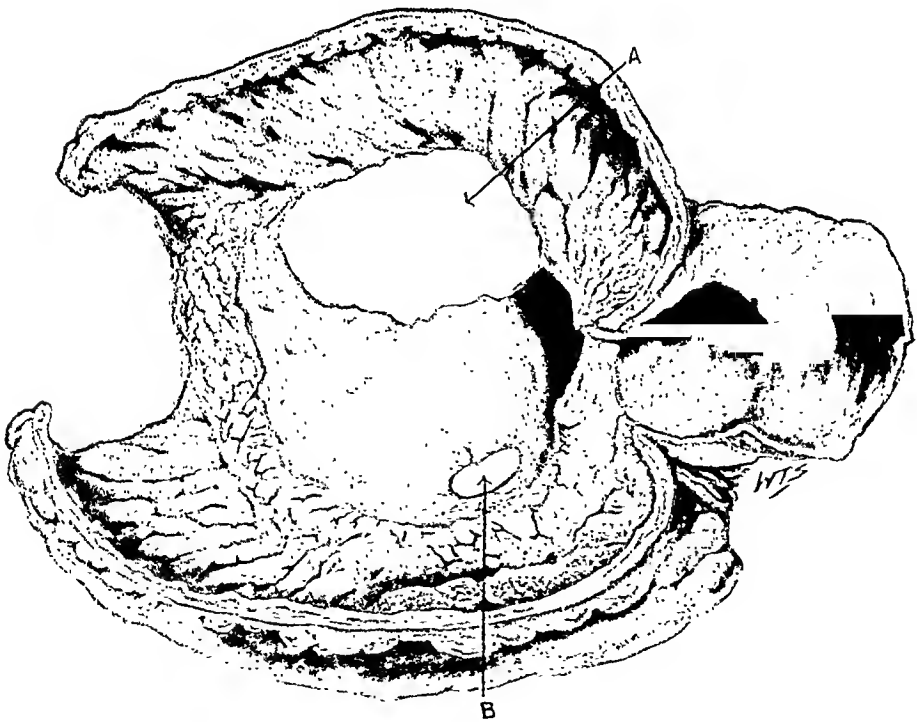


FIG. 234.—Same case as *Fig. 233*, showing: A, Posterior perforation; B, Perforation into anterior abdominal wall.

completely eaten through the posterior wall of the stomach, its floor being composed of pancreas and mesocolon.

As is commonly the case, the lesser curvature has been so shortened by contraction that an acute kinking of the pylorus has been produced, and the gastric stasis is due to this kink and not to a true stenosis. Partial gastrectomy by the anterior Pólya method was done, and the patient is now, five and a half years after the operation, in good health and able to lead a normal life. This was a patient in whom any form of gastro-enterostomy would have been extremely difficult, if not impossible, as there was very little healthy gastric wall except at the extreme cardiac end of the stomach.

Since that time I have employed partial gastrectomy in all massive and complicated ulcers which have come under my care. On two occasions I have found a small abscess cavity between the stomach and liver, the result of chronic perforation ulcer, and, in spite of this, partial gastrectomy has been successfully carried through.

In one case of hour-glass contraction of the stomach in a woman, age 46, on whom I operated in January, 1925, on opening the abdomen the upper sac was found to be quite hidden under the ribs, by adhesions of an active ulcer to the anterior abdominal wall. The appearance was so unpromising that I contented myself with gastrostomy into the distal sac, in the hope of giving the patient relief and allowing her general condition to improve. Her pain persisted and her general condition did not improve. On March 26, 1925, I operated again, and by separating the adhesions from the anterior abdominal wall, the liver, and the pancreas, I was finally able to free the stomach and do a Pólya resection by the anterior method. A very small portion of stomach remained, but in spite of this the patient is now quite well. The cure is so dramatic and complete that I have no hesitation in adopting this treatment. As will be seen shortly, the risk is not very great.

TECHNIQUE OF OPERATION.

There are a few points in the technique of operation in these extensive cases that are of importance. It is useless to expect to deal with such large ulcers without opening the stomach and soiling the peritoneum to some extent. Indeed, I believe there are special risks in attempts to keep the floor of the ulcer intact by taking slices off the pancreas. It is therefore important to wash out the stomach thoroughly for some days before the operation. This should only be omitted if there has been a fairly recent hæmorrhage. After separating adhesions to the anterior abdominal wall, I divide the great omentum along the greater curvature of the stomach. In this way the stomach can be lifted up and the gastroduodenal artery is easily exposed and divided between two ligatures. The lesser omentum is then perforated near the pylorus, and the pyloric artery similarly treated. The posterior wall of the duodenum is stripped as far as necessary from the pancreas, two crushing clamps are applied to it, and it is divided between them. The distal end of the duodenum is then closed by suturing over the clamp by the method of Moynihan. The stomach is now pulled over to the left, and it becomes easy to define the adhesions to the pancreas, mesocolon, and liver. Where the ulcer is of large size no attempt is made to preserve

the continuity of the stomach wall. This is separated from the pancreas and mesocolon where it joins them, the gastric cavity being entered. The stomach is detached in this way all round the margins of the ulcer; and separation from the liver is, if necessary, done in the same manner. The floor of the ulcer, which is left behind on the pancreas, is then gently curetted and treated with pure carbolic acid. If the peritoneum is carefully packed off, this proceeding is perfectly safe, and is preferable to the removal of a slice of pancreas. This latter manoeuvre is recommended by some surgeons in order to keep the stomach intact and prevent soiling. In addition to being impracticable in very large ulcers it carries a special risk of its own—pancreatic fistula.

In one patient with an hour-glass contraction of the stomach and an active ulcer adherent to the anterior abdominal wall, the liver, and the pancreas, I shaved off a piece of pancreas, as the adhesion to the pancreas was not more than an inch in diameter. A few days after operation the wound broke down and large quantities of a clear watery fluid came away. This was strongly alkaline and contained a sugar-inverting and a proteolytic ferment, and was quite clearly pancreatic juice. The fistula persisted for nearly six months before it finally closed.

In order to understand how this happened it is necessary to study the anatomy of the pancreatic ducts. The main duct commences at the tail and runs along the gland, nearer the upper than the lower border. It receives lateral tributaries along the whole length of its course, the arrangement being known as a 'herring-bone' type. I tried to inject the pancreatic ducts in order to see the relationship to the surface, but all attempts failed, the duct walls giving way under the pressure of the injecting fluid. In Poirier and Charpy's *Anatomy*, however, I found a photograph of an injected specimen by Marie, in which it can be seen that some of the tributary ducts are of large size, even to the surface of the gland. It is easy to understand how a large duct might be entered by taking off even a thin shaving of glandular tissue and a fistula result. It is preferable, therefore, to detach the stomach in the manner described.

After the stomach has been freed in this way it only remains to secure the coronary vessels and the vessels on the greater curvature at the level at which we decide to make the stomach section. The coronary vessels are best secured as they run forwards from the posterior abdominal wall, as already noted in describing the mobilization of the lesser curve in local resections of the ulcer. The jejunum is next brought in front of the colon, just sufficient loop being used to avoid compressing the colon, and is united to the stomach, the posterior layer of seromuscular sutures being inserted before the division of the stomach is accomplished. Clamps are used, both on the stomach and the jejunum, and the jejunal loop runs from the greater to the lesser curvature of the stomach.

RESULTS OF GASTRECTOMY.

I have employed partial gastrectomy in the treatment of chronic gastric ulcer on 63 occasions. Some of the early cases were by the Billroth II classical method, and others by the posterior Pólya method or the anterior

method with the jejunal loop running from small to great curve. The great majority, however, are by the modified method I have described. Three cases died as the result of operation. One patient, who had a very large saddle-shaped ulcer and was much reduced by pain and starvation, died of shock within twenty-four hours. One, a man of 54 years, had a large ulcer penetrating the pancreas over an area one and a half inches in diameter. He died of heart failure on the fifth day, although up to that point he had been progressing favourably. The third was a remarkable case of a woman of 49 years, with a history of many years of gastric pain and with increasing constipation and vomiting for three months. She was very much emaciated, and the abdomen was greatly distended, with large peristaltic tumours in both flanks and a 'ladder pattern' peristalsis in the middle of the abdomen.

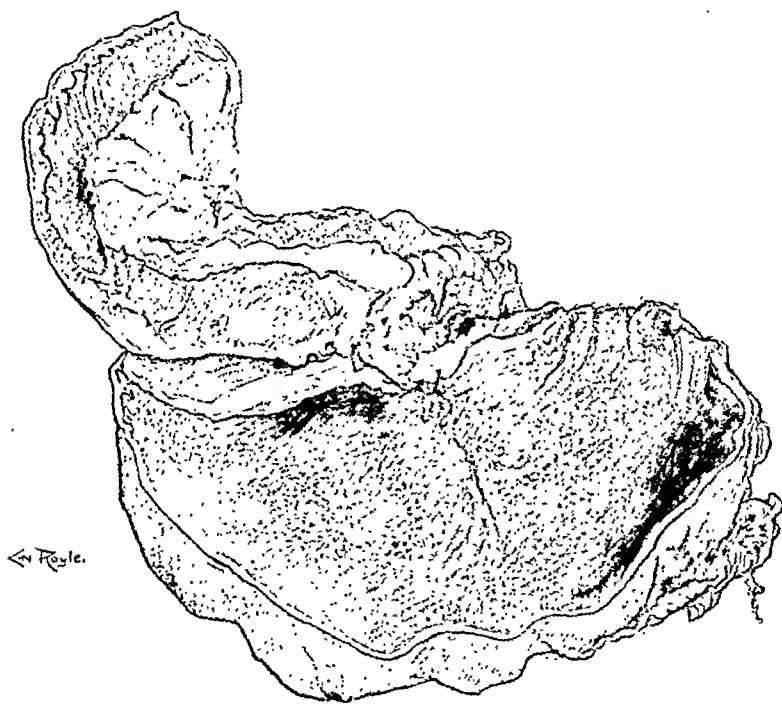


FIG. 235.—Gastro-ileal fistula.

I operated on Feb. 25, 1921, and found an enormously distended stomach. There was a congenital opening in the mesocolon through which the small bowel had prolapsed. It had then prolapsed through the lesser omentum over the lesser curvature of the stomach so that the terminal ileum was lying across the anterior surface of the stomach. At the point where the ileum was in contact with the lesser curve there was a gastric ulcer which had perforated into the ileum, a fistula resulting (*Fig. 235*). In addition, the ileum was obstructed at this point, so that I was faced with a complicated problem. I resected the ileum and did a lateral anastomosis. I then reduced the small gut through the abnormal openings and closed these, and finished up with a Pólya partial gastrectomy. At the time I thought I was dealing with a

carcinoma, but it proved not to be so. The patient died of shock sixteen hours later.

If we exclude this case it leaves us with 2 deaths in 62 cases of partial gastrectomy for chronic gastric ulcer. This series does not include operations undertaken for the arrest of acute hæmorrhage from ulcer, of which I have 3, with 2 deaths. Such cases must be classified by themselves, as they are desperate efforts to save life in an acute emergency.

As regards the remote results, I have been able to trace 44 of the 60 survivors of the operation. Four of them have died since the operation—one from meningitis, one from phthisis, one from carcinoma, and one became insane and died in an asylum. The death from carcinoma took place about a year after operation, and in all probability the operation diagnosis was incorrect, although it was confirmed by microscopic examination.

One patient developed regurgitant vomiting for which entero-anastomosis was subsequently necessary. This was a fairly early case, and I used an excessively long jejunal loop. Food regurgitated into the duodenal end of the bowel and accumulated there, relief being obtained by attacks of vomiting. The patient has been greatly relieved by the entero-anastomosis, but still has pain occasionally.

One other patient has vomiting about once a fortnight, and radiographically food can be seen passing into the duodenal end of the bowel. Otherwise he is in good health and takes all kinds of food. Two others say that very occasionally they have very slight vomiting. The remaining 36 patients are in excellent health and live ordinary lives.

From these results it would seem that the only unsatisfactory results are due to a technical defect in the anastomosis which allows the food to pass into the blind end of the bowel, where it accumulates, relief being afforded from time to time by attacks of vomiting. The risk of this can be minimized by making the section of the stomach well towards the cardiac end and by using as short a loop of jejunum as possible without compressing the transverse colon. When one considers the extent of the lesion in most of these patients, and the pain and suffering which afflicted them, I submit that the results of the operation fully justify it.

REFERENCES.

- ¹ DIBLE, J. H., *Brit. Jour. Surg.*, 1925, xii, 666.
- ² PERMAN, E., *Acta Chir. Scand.*, 1927, lxi, 465.
- ³ HARTMAN, *Chirurgie de l'Estomac*.

ELEPHANTIASIS.

By A. P. BERTWISTLE,

MEDICAL OFFICER TO THE LONDON DISPENSARY, SPITALFIELDS;

AND A. L. GREGG,

LATE MEDICAL SUPERINTENDENT, HOSPITAL FOR TROPICAL DISEASES, ENDSLEIGH GARDENS.

By reason of its hideous deformity, elephantiasis has always interested not only medical men but also the lay public. Its causation has for long been a matter of dispute and, although over eighty out of some ninety references have been traced, the literature makes rather confusing reading.

It is a disease of great antiquity, but we have failed to find any record of it in Egyptian or classical times. In the sixteenth century the Portuguese must have brought back tales of the long rows of sufferers who, then as now, line the bridge leading to the shrine of Kali in Calcutta. The first recorded case of non-filarial elephantiasis was that of Kennedy¹ in 1796, which resulted from sepsis following the bursting of a matchlock; Kennedy had seen the tropical form in Madras. In 1817 Chevalier² reported one case following phlegmasia dolens. Southam,³ in 1902, described the condition in a lady, of which a quaint woodcut was published.

This disease is common in those parts in which *Filaria bancrofti* is prevalent, less frequent in other tropical and subtropical places. It is found sporadically in the British Isles and France, less commonly in Germany and Austria, where it probably affects men more than women, the lower limbs—usually the left—being involved.

DEFINITION.

Matas⁴ defines elephantiasis as “a progressive histopathological state characterized by chronic inflammatory fibrosis and hyperplasia of the dermal and hypodermal tissues, which is preceded by and associated with venous and lymphatic stasis.” Unna⁵ considers that elephantiasis develops in the œdema which results from repeated attacks of erysipelas. Hutchinson⁶ describes it as “a condition resulting from solid œdema and more or less resembling the appearance of an elephant’s limb.” He goes on to state that “although this condition is much commoner in the tropics than in temperate climates it occurs in the latter with features as definite as in the former.” He was convinced that elephantiasis did not occur unless there had been attacks of elephantiasis, or, as it is more generally called, ‘elephantoid’ fever.

Whatever definition is adopted, it is very necessary to distinguish elephantiasis from lymphatic œdema, a large number of cases published under the name elephantiasis being really examples of solid œdema in which there was no hypertrophy, but merely a distention of the cells and spaces.

Undoubtedly, true elephantiasis is a condition of hypertrophy and

hyperplasia of the skin and subcutaneous tissues formed in a part suffering from lymphatic and, probably, venous obstruction as a result of bacterial infection, usually streptococcal.

Elephantiasis nostras is the term applied to the condition when occurring in temperate climates, as distinct from *Elephantiasis tropica*, which includes *E. arabum* (filarial) and *E. græcorum* (leprol). *E. neuromatosis* is a form of von Recklinghausen's disease, a condition "congénitale toujours, héréditaire souvent, et quelquefois familiale" (Feindel), which has an altogether different pathology.

BACTERIOLOGY.

In 1893 Sabouraud⁷ found in his five cases of elephantiasis that, by scarifying the skin during the inflammatory stage, he was able to grow a pure culture of Fehleisen's streptococcus, but that during the quiescent period they were not to be found.

Prout⁸ found the same thing in the tropical form. Dubreuilh⁹ considered the patient infectious during this stage. Elliot¹⁰ was able to demonstrate, by means of Unna's polychrome methylene blue method, the presence of clumps of cocci round the vessels beneath the cutis during, but not after, a febrile attack. We find no reference to a method now in use—namely, the injection of saline intradermically and its withdrawal some time later for culture.

That cocci can live in symbiosis with the tissues as suggested by Hutchinson⁶ is more than doubtful, though they may long remain latent, as was made manifest in the Great War.

The probability is that the cocci are from time to time borne by the blood-stream and settle in a 'locus resistentiæ minoris' caused by some circulatory disturbance either hæmic or lymphatic. The focus of origin may be a local wound or ulcer, or some distant site such as the teeth (*Case 1*) or the tonsils, as in two of Sistrunk's cases.¹¹

The repeated occurrence of the condition in one or more areas is readily explained by analogy with erysipelas, which renders the patient and the same site more liable to further attacks. Bockhart¹² succeeded in obtaining the experimental production of elephantiasis by inducing repeated attacks of erysipelas.

HISTOLOGY.

This has been carefully studied by Alberca,¹³ Elliot,¹⁰ and Unna.⁵ Unna describes two stages: "The first, or soft, stage is characterized by active subcuticular metamorphosis of certain areas, this being the later result of streptococcal infection which primarily causes atrophy: the connective tissues form plasma cells which are seen throughout the skin and hypodermis. These cells are encased in collagenous material which is greatly increased in bulk and arranged in coarse bundles parallel to the skin, maximal at the margin of the cutis, in such great amount that it distorts the plasma cells, which become multinuclear. Large ovoid mast cells are seen here and there; elastic tissue is absent.

"The second, or hard, stage is manifested by a still further increase in

collagenous material, but the soft swelling gives place to hyperplastic tissue. The surface epithelium first shows hyperplasia, some cells resembling those found in condylomata; later it retains its thickness only by hyperkeratinization; finally, mottled pigmentation occurs."

Alberca states that the skin is thinned before it is thickened. In one case he found the plasma cells degenerated, containing vacuoles which did not give the reactions of fat, colloid, or mucus; in another case he discovered long crystals in the fibroblasts. Differing from Unna, he found, in the neighbourhood of the epidermis, an increase in elastic tissue, which, like that of large arterics, formed a fenestrated membrane.

PATHOLOGY.

Hutchinson⁶ laid down that there could be no elephantiasis unless venous and lymphatic obstruction were associated with streptococcal infection, and that whether the disease occurred in tropical or temperate climates the *essential* pathology was the same.

There has been much unnecessary controversy as to whether the œdema which precedes the condition is caused by venous or lymphatic obstruction: for the development of solid œdema both are essential, as has been proved time and again experimentally. The intimate association of the venous and lymphatic circulations is strikingly shown by the great increase in lymph flow which occurs in a limb when its vein is ligatured. In carcinoma of the breast no œdema develops unless both the axillary vein and lymph channels are affected.

The cause of the venous obstruction is usually a thrombosis. Chevalier's case² followed phlegmasia alba dolens; Wintrop¹⁴ described an excellent case which resulted from the same condition after attacks of erysipelas. Thrombosis may occur after infections such as typhoid fever, influenza, or pneumonia (cf. Case 2).

The causes of lymphatic obstruction are many, and may be classified as follows:—

1. **Congenital.**—There is a condition first described by Milroy,¹⁵ and independently by Meige, in which there is an hereditary maldevelopment of the lymphatic vessels showing itself as œdema. Hope and French¹⁶ describe an excellent case of hereditary swollen leg in which elephantiasis developed as the result of streptococcal infection with thrombosis.

2. **Traumatic.**—Under this heading must be included those cases of œdema resulting from extensive dissections of glands, probably associated with cicatricial contraction of connective tissue leading to narrowing of the lumen of the vein. There is recorded in the literature one case of elephantiasis developing after a crush. Formerly, a recruit for the army was rejected if swollen inguinal glands were present, so these were sometimes dissected out: a certain number of the men so treated developed œdema, and in some of these elephantiasis followed (cf. Case 3).

3. **Infective.**—There are a number of organisms and parasites which affect the lymphatic vessels and glands; the following may be mentioned:—

- a. *Filaria bancrofti* is by far the commonest predisposing cause of

elephantiasis, but has wrongly been considered the only cause. The adult worms live in the lymphatic system, and the embryos pass into the general blood-stream via the lymphatic vessels. It is probable, however, that the presence either of the adults or of the microfilariae cannot in itself cause lymphatic obstruction; this is rather the outcome of secondary sepsis, organisms occasionally present in the blood-stream invading the dead helminth and devitalized tissues. Thus the predisposing cause of the resultant elephantiasis is the filarial infection, the exciting and essential cause is the secondary septic infection.

b. Tubercle bacilli are well known for their affinity for the lymphatic glands. The condition of tuberculous elephantiasis arises as the result of lupus or tuberculous ulceration and concomitant infection of the lymphatic vessels. Such a case has been well described by White.¹⁷

c. Syphilis is a not infrequent cause of lymphatic obstruction, upon occasion being followed by elephantiasis (Sequeira¹⁸ and Adamson¹⁹).

d. Leprosy occasionally causes elephantiasis through an invasion of the lymphatic system by the *B. lepræ*, the condition being known as *E. græcorum*. Secondary infection from local sores is almost invariably present.

e. Granuloma venereum or *inguinale* is stated to cause sometimes an elephantoid condition of the labia minora as the outcome of sepsis and scar formation.

f. Infective lymphangitis, of whatever source, is undoubtedly by far the commonest cause of lymphatic obstruction. Halstead states that it is the cause following breast operations.

g. Malignant disease can readily cause œdema by means of carcinoma cells encroaching upon the veins and lymphatic vessels, but the condition can rarely be sufficiently chronic for elephantiasis to develop. In a personal communication Handley mentioned that he had never seen a case of elephantiasis develop from mammary cancer. There is, however, one striking case of elephantiasis arising out of cancer of the prostate described by G. Carmichael Low²⁰. One of us (A. L. G.) was associated with this case, and performed the autopsy. The thickness and density of the corded pelvic and lumbar lymphatics was astonishing.

h. Toxic.—It is convenient under this head to mention a case encountered by Manson-Bahr of elephantiasis following the absorption of chrysarobin in the treatment of psoriasis.

That another factor besides lymphatic and venous obstruction is necessary for the development of elephantiasis is suggested by: (1) The presence of solid œdema for long periods without the appearance of elephantiasis; (2) Its development in cases of solid œdema after attacks of fever and lymphangitis.

It appears necessary, therefore, that not only must there be a transudate of lymph, but that it must be changed in some way, as happens when it becomes an exudate as the result of inflammation. It has been shown that the lymph exudate in cases of elephantiasis is charged with albumin, and, as protein is a stimulant to cell activity and growth, the key to the hypertrophy and hyperplasia probably lies in Starling's²¹ description of the function of lymph: "The only way the tissues can receive their supply of protein is

from the small amounts which are filtered through the blood-vessels into the lymph. The increased exudation of concentrated lymph which occurs in inflammatory conditions as the result of injury is therefore of advantage, since it furnishes an abundant supply of protein food to be used up in the regeneration of the damaged cells." In elephantiasis the affected part receives an increased supply of protein as the result of recurring attacks of inflammation, and it is suggested that this protein, instead of merely helping in the process of repair, serves by its continuous or perverted action to stimulate the connective tissues to excessive growth.

As regards the infecting organisms, the origin of the streptococci may be from a local or distant focus, and the tropical form of elephantiasis has given rise to much controversy. Some authorities contend that a specific organism causes the lymphangitis, which in turn kills the filaria; but it appears more reasonable to suppose that the death of the parasite occurs first, and its body becomes a nidus for the development for such cocci as may be carried to it by the blood from any septic focus in the body. Once infected, the worm's body serves as a local reservoir or focus for recurring infection of the lymphatics and veins.

The precarious life of the parasites and the prevalence of septic foci make elephantiasis in filarial countries a disease of great frequency; whereas the rarity of the combination of venous obstruction, lymphatic stasis, and streptococcal infection accounts for the small incidence of the disease in non-filarial countries.

CLINICAL FEATURES.

Elephantiasis, when well developed, is sufficiently characteristic to require little description, but the early stages are not so easily recognized in non-filarial conditions, although only too well known and dreaded where filarial elephantiasis abounds.

The Site.—Elephantiasis may occur on any part of the body, being reported of scalp, face, tongue, breast, vulva, penis, testes, and buttocks, where it may take the pedunculated form; all these, however, are unusual, the legs, serotum, and arms being most commonly affected. *Table I*, showing the relative frequency of the parts attacked, is compiled from 4712 cases of tropical elephantiasis reported by various authorities:—

Table I.—RELATIVE FREQUENCY OF PARTS ATTACKED IN 4712 CASES.

LEGS	SCROTUM	ARMS	LEGS AND SCROTUM	LEGS AND ARMS	LEGS, ARMS, AND SCROTUM	BREAST
Per cent 57	Per cent 38	Per cent 0.9	Per cent 0.9	Per cent 3.5	Per cent 0.8	Per cent 0.1

The vulva is reported as being affected in only 0.8 per cent of cases, a figure, from the natural reticence connected with the condition, probably much too low. With tropical elephantiasis, however, the parts most often affected vary with the country; thus the arm is affected in 42.6 per cent of cases in Fiji, yet scarcely at all in the French Congo.²²

In non-filarial elephantiasis the leg is the most commonly affected part, although it has been reported of the face (Mitchell²³), scrotum (Low²⁰), and is here recorded of the labia (*Case 2*), penis and scrotum (*Case 3*), arm and legs (*Case 4*).

The Onset.—In a few patients some degree of oedema may gradually occur in a part, but the actual commencement of true elephantiasis always dates from the first attack of lymphangitis, whether or not there has been preliminary oedema. The initial lymphangitis is sudden, frequently occurring during or shortly after an acute illness such as tonsillitis, pneumonia, etc., and becoming manifest either as a phlebitis or erysipeloid attack with localized lymphatic inflammation. These phenomena are associated with considerable constitutional disturbance, such as sudden pyrexia, anorexia or vomiting, and general body pains. During the first attack the diagnosis of a local inflammation is alone possible, but should a slight residual thickening occur followed by similar attacks, it will permit a diagnosis of commencing elephantiasis to be made, and precautions should be taken forthwith to ward off further inflammatory attacks leading to unsightly enlargement.

Progress.—The progress of the disease varies in its chronicity, and may be described in three stages which insensibly merge into each other.

First Stage.—The first stage is evidenced by smooth uniform swelling or thickening of the part, the overlying skin being smooth, pale, and cool, except during attacks of lymphangitis. Apart from such attacks, little disfigurement or discomfort is caused; the attacks may occur at short or long intervals.

Second Stage.—A second stage may be described when the skin shows definite thickening and coarsening, and acquires an uneven, ridged appearance, and the muscles connected with the part are hypertrophied to compensate for the increased burden.

Third Stage.—In the third or fully developed stage the skin and subcutaneous tissues are greatly hypertrophied; in the case of a limb they are thrown into vast folds separated by deep sulci occurring not only at the flexures but elsewhere, while in the scrotum there is diffuse ruggedness rather than these definite divisions. Upon occasion the skin is thrown up into bosses or numerous wart-like projections (*E. verrucosa*), while at all times there is the liability to weeping fissures, indolent ulcers, or abscess formation. If the ankle and foot are swollen and the toes unaffected, the peculiar 'tortoise-foot' appearance is seen. At this stage the muscles show some degree of pressure atrophy, and slight bone distortion may be observed. As the disease progresses, the intervals between the attacks of lymphangitis may become prolonged until the attacks cease altogether, or the attacks may increase in number and severity, the lymphangitis extending more and more proximally until deep abscess formation and septicæmia cause death.

Contrary to what might be expected, the blood-supply of the part is usually good, the veins becoming enormously enlarged. It is probably because of this that foreign solutions such as a dye are absorbed from elephantoid tissue in normal time, as was demonstrated from the renal excretion in *Case 2*.

Elephantoid tissue does not pit readily, but firm prolonged pressure will cause an indentation. This has a practical application in the use of a tourniquet applied over elephantoid tissue before an operation: when first tightened the tourniquet may be efficient; later, indentation occurs beneath it sufficient to permit arterial flow and still prevent venous return, so causing profuse hæmorrhage. If, then, it is desired to use a tourniquet at all, it is advisable before starting the operation to place around the part a second tourniquet against the need of additional tightening.

TREATMENT.

Focal Sepsis.—In the treatment of elephantiasis attention must first be directed to sources of focal infection, as the attacks of fever and lymphangitis are due to recurring infection from some septic focus, and the removal of this, Mr. Eccles tells us, may produce an amelioration of even an advanced condition. The result of an operation is imperilled if this focus is not treated beforehand. For example, in *Case 1* the septic foci were infected teeth, while in two of Sistrunk's¹¹ patients the tonsils were diseased. Though teeth and tonsils probably form the chief source of infection in non-tropical elephantiasis, appendicular, pelvic, or urinary infections may be responsible.

General Health.—The second consideration is to improve the patient's general health and powers of resistance. Rest in bed is necessary during the early part of treatment, and endeavour should be made to encourage the patient and improve his nutrition.

Vaccine therapy has a definite place in treatment, and is discussed at length in the report of the British Guiana Commission.²⁴ Their conclusions are that vaccines frequently abort further attacks of fever and improve the chances of recovery after operation. An autogenous vaccine is best when obtained, as may often be done, by the intradermal introduction of saline into the affected area during an attack of fever with a view to withdrawing the fluid shortly for culture. *Serum* has also been used with marked success; Matas⁴ advises frequent injections of polyvalent antistreptococcal serum. Specific treatment is, of course, indicated when the disease is the outcome of, or accompanied by, syphilis or leprosy.

Local Conditions.—Elevation of the affected part is essential throughout treatment, and those suffering from elephantiasis of the lower limbs should sleep henceforth with the foot of their bed slightly raised. In the daytime such patients should have the affected limb supported by constriction, as may be done by a crêpe bandage, elastic stockings (made to lace up), or the less expensive 'Occulta' stockings.

Massage of the affected part in the absence of inflammation will do much in restoring the vitality of the affected tissues; it should be deep and vigorous.

Such measures may appear commonplace enough, but their proper application suffices to arrest mild cases, and if applied some weeks before operation permit a much better prognosis. The need for this preparation is emphasized by Sistrunk,¹¹ and from our experience we readily endorse his opinion. It will be found that the healing of ulcers of long duration can thus be achieved.

Operations.—When, and only when, the foregoing measures have proved insufficient, must operation be considered. At one time amputation was the only operation practised, and it remains so for scrotal and breast enlargements. For the limbs, the stages by which we have come to regard the Kondoléon operation as the ideal one are interesting. Conservative treatment was first attempted in 1851, when Carnochan²⁵ ligatured the femoral artery in the hope of starving the growth. Based on wrong pathology, this was soon abandoned. In 1908 Sampson Handley²⁶ published his work on lymphangioplasty. The method was successful in relieving oedema of the arm resulting from cancerous deposits, but, as would be expected in a disease whose background was a streptococcal infection, the silk caused irritation and the channels became occluded. In his own words, "This hope that lymphangioplasty might establish for itself a definite position in the treatment of elephantiasis seems to be doomed to disappointment."

In 1911 Lanz²⁷ reported a cure by embedding multiple strips of deep fascia into trephine openings in the bone, after first trying the effect of interposing them among the muscles. Later he suggested implanting the spermatic cord with its rich plexus of lymphatics into the inguinal region. This was accidentally done by Milroy, who, by excising a diseased testis, brought about the cure of a leg affected with the disease which bears his name. In 1912 Kondoléon,²⁸ a Greek, published seven cases which form a landmark in the treatment of elephantiasis. As long ago as 1874 Sappey²⁹ had pointed out that the deep fascia forms an impenetrable barrier to the exchange of lymph between the superficial and deep lymphatic systems. Kondoléon confirmed this statement clinically by observing that the thickening in elephantiasis was limited to the skin and subcutaneous tissues. His curative principle, therefore, was to remove long strips of deep fascia and so establish communication between the deep and superficial lymphatics.

The technique for the lower limbs as slightly modified by Sistrunk³¹ is as follows: An external incision is made from the external malleolus to the great trochanter of the femur, and an internal one from the internal malleolus to the mid-Poupart line, no attempt being made to preserve the internal saphenous vein. A large slice of oedematous skin and subcutaneous fat is removed, and the aponeurosis removed three fingers in breadth throughout the length of the incision. The skin is then sutured in contact with the muscles, leaving no provision for drainage. The operation is conducted in stages. The wounds were found to heal readily, a point in favour of the view that there are no streptococci lying quiescent in the tissues but that they are intermittently deposited there. Later Sistrunk advised removal of redundant skin. He reports 31 cases with excellent results. Hill,³⁰ Royster,³¹ and Henry³² each report a successful operation.

In dealing with scrotal enlargements the operation may be complicated by the presence of a hernia, hydrocele, abscess formation, etc. Careful preliminary examination and preparation are essential if disaster is to be avoided. Amputation, with special measures for ensuring lymph drainage, is advisable, a new scrotum being formed, if necessary, by plastic methods. Hill³⁰ describes such an operation: "The incisions were made at the external abdominal rings, the cords were found in a mass of blubbery areolar tissue. I

Esmarched the scrotum and removed it entirely, with the loss of little blood. Bringing the organs into position I covered them with the adjacent skin, and in the extensive opening up I was careful to expose as much as possible of the deep muscular spaces. Upon the dorsal surface an incision was made the whole length of the penis, which was freed from its covering. A very extensive dissection of the skin over the pubis enabled me to sew the pubic skin to the mucous membrane of the gland, and it practically covered the organ. In this case an attack of elephantoid fever ensued, so Matas' serum treatment was applied. The end-result was eminently satisfactory."

This is quoted as a concise outline of the operative procedure, but we follow Chatterji³³ in discarding the use of a tourniquet altogether and dividing the base of the tumour as the first step, so exposing and clamping the chief vessels as they converge upon the scrotum. Chatterji³³ advises against the use of pubic skin as a covering for the penis, on the grounds of thickness and hairiness: he prefers to leave a raw penile surface to which skin-grafts may later be applied (cf. *Case 5*).

PROGNOSIS.

As regards life the progress is usually very fair, although in the tropics many sufferers from elephantiasis die as the result of acute septicæmia following an unusually severe attack of lymphangitis. In the late stages of the disease patients may be bed-ridden with deformity and occasional pain. For the limbs, when the condition is advanced, the Kondoléon operation holds out the best prospects of a cure, though it is probable that the fibrous covering of the muscles will eventually be replaced. This actually occurred in *Case 2*, as was proved during the fifth operation by finding that a thick fascia, indistinguishable from the original deep fascia, was present covering muscles from which it had previously been removed. Nor was there any sign of muscle hernia as suggested by Henry.³² On the permeability of this fascia is said to depend the success of the operation; probably it is only a matter of time till it becomes so thick as to be impermeable to the lymph.

CASE REPORTS.

Case 1.—Male, never resident in the tropics, age 27 in 1923, was recovering from an acute attack of tonsillitis when pains started around the left ankle, succeeded next day by local œdema and 'tightening pains' in right chest accompanied by fever. The œdema lessened but never disappeared, being kept in check by a crêpe bandage. Two months from the onset the tonsils were enucleated; five months later an acutely inflamed appendix was removed. Other systems normal. Good health for four years: then began a series of attacks of elephantiasic fever. The first started with sudden fever followed by profuse vomiting. Twelve hours later the superficial inguinal glands and then the whole course of the lymphatics accompanying the internal saphenous vein became acutely inflamed, though the vein itself was unaffected. In a month the leg was normal except for an increase of œdema. Similar attacks occurred during the next three years, vomiting becoming less noticeable. Sometimes fever was the first sign, at times the glandular swelling, at others inflammation of the lymphatic vessels. Definite circumscribed areas of skin were always affected, becoming a dusky-red colour, œdematous, raised, and desquamating freely after the attacks. A dental septic focus was suggested by Mr. McAdam Eccles although no obvious caries was present. The second left molar was bluish in colour;

it had been filled, but its root not treated; skiagrams disclosed a root abscess. An attempt was made to sterilize the abscess, but a mild attack of fever recurring three months later, the tooth was extracted by Dr. F. O. Human. One of the roots appeared almost broken, so great had been the absorption. Bacteriological investigation was unsuccessful owing to the previous antiseptic dressings. Mild attacks of fibromyositis, which had formerly been present, disappeared after the extraction. (The case has been described elsewhere³⁴ up to this stage, though its nature was unknown.)

One year later a somewhat atypical attack occurred, and two days later a crowned tooth became tender and fibromyositis again appeared. Skiagrams showed nothing abnormal; the tooth, however, was extracted, and a granuloma was found on one root. Bacteriological examination by Dr. R. Canti disclosed streptococci and diphtheroids in the granuloma. The pulp cavity was full of greenish pus from which a scanty growth of *Staphylococcus albus* was obtained. This second extraction was done on Aug. 27, 1927; since then there have been no further attacks.

COMMENTS.—The primary lesion was clearly a thrombosis of the venæ comites of the posterior tibial artery as a complication of the tonsillitis. The series of febrile attacks was the result of inundation of the damaged limb with showers of streptococci periodically liberated into the blood-stream from the septic teeth. Inattention to these septic foci would undoubtedly have led to elephantoid tissue supervening upon the œdema at the left ankle.

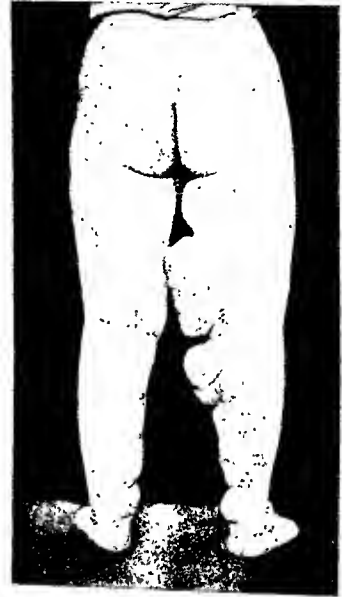
*Case 2 (Figs. 236-246).—*An unmarried woman, age 31, was admitted to the Hospital for Tropical Diseases on Feb. 4, 1924, under Dr. G. Carmichael Low.

HISTORY.—Family healthy except one brother who died two years previously from phthisis. Previous illness unimportant. Present illness began in 1907, at the age of 14, with painless gradual swelling near the right ankle without apparent cause when the patient was in good health. About 1912 the left ankle began to swell in a similar manner. In 1917 she was confined to bed for two months with pneumonia and inflammation of both legs. Delirium was present for some days. Four successive abscesses formed and burst spontaneously in the right leg. This illness was followed by a more rapid increase in the size of the limbs and recurring attacks of lymphangitis, always in the right leg except the last two, which affected the left leg. These attacks occasioned great pain, and the last occurred a fortnight before admission. Both legs at times exude a clear fluid through small holes, these appearing one at a time and closing spontaneously in about a week.

PHYSICAL EXAMINATION.—General nutrition good and systems all normal except for a rather nervous temperament suggestive of slight hyperthyroidism. Teeth carious. Abdominal palpation, rectal and vaginal examinations, and cystoscopy disclosed nothing abnormal, nor did radiograms of the pelvis and abdomen. Blood-counts: red cells, 4,550,000; white cells, 12,000; hæmoglobin, 80 per cent; polymorphonuclears, 69 per cent; lymphocytes, 27 per cent; large mononuclears, 3 per cent; eosinophils, 1 per cent; no parasites. The labia majora were elephantoid and warty in appearance, left worse than right; rest of vulva normal. Both lower limbs were greatly enlarged below the inguinal and buttock flexures (Figs. 236 and 243). Chronic inflammatory changes in skin above both ankles and on postero-internal aspect above right knee.

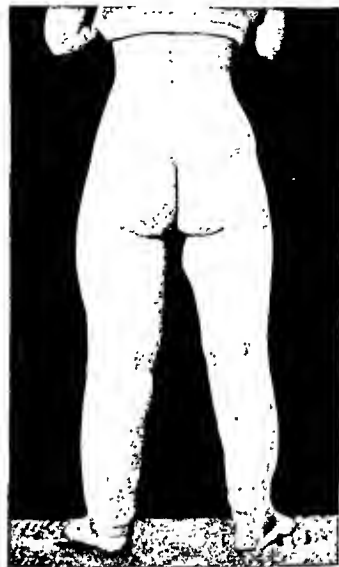
PROGRESS.—After a fortnight's preliminary cleansing, massage, and dental treatment, operative measures were started, very little being done at first on account of the short time that had elapsed since the last febrile attack. Colonel J. J. Pratt was in charge of the first three operations, in association with one of us (A. L. G.), who carried out the remaining four operations, the patient being allowed home between some of them. The last operation was performed on Oct. 25, 1924, practically all of the elephantoid tissue on the limbs and labia having been removed; Figs. 237 and 238 give some idea of the intermediate stages. The operations consisted of removal from below upwards of as much elephantoid tissue as possible by means of elliptical incisions and undermining, thus removing large masses of blubbery tissue, including underlying portions of deep fascia. The incisions were planned so that the skin edges could best be drawn together. A tourniquet was used for the first operation only, being found unsatisfactory owing to the œdema of the thighs. The

redundant tissue of the labia was removed in a similar manner. Tension sutures when used were removed on the third to the fifth day, some of the remaining sutures being left to the tenth or twelfth day. Primary union was obtained on each occasion.



FIGS. 236, 237, 238. —Case 2. Indicating the stages of operation.
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Since 1924 the patient has been seen from time to time, and has remained in good health, free from severe lymphangitis, though attacks of a very mild character occasionally occur. Her activity is nearly normal; she has even danced! The



FIGS. 239 and 240.—Case 2. Showing results about two and four years later.



FIG. 241.—Case 2. Side view. Note that indentations are not confined to the flexures.



FIG. 242.—Case 2. Nearly four years later.

scars are all soundly healed, nor is there ever any weeping or oozing from the limbs; the new-formed labium majus has slightly increased in size, and the labia minora are a very little enlarged. The patient was last examined on Jan. 22, 1928, and the measurements then taken are tabulated for contrast with former measurements, while Figs. 240, 242, and 244 show the present condition.



FIG. 243.—Case 2. Before treatment.
Note labial swelling.



FIG. 244.—Case 2. Almost four
years later.



FIG. 245.—Case 2. Photographed for comparison with a normal woman of about same age and height.



FIG. 246.—Case 2. Photographed with the same woman nearly two years after treatment.

Table II.—CIRCUMFERENTIAL MEASUREMENTS IN INCHES IN CASE 2.

POSITION OF MEASUREMENTS	RIGHT LEG			LEFT LEG		
	1924 Feb. 4	1924 Nov. 11	1928 Jan. 22	1924 Feb. 4	1924 Nov. 11	1928 Jan. 22
At top of thigh	23½	22½	20	22	22½	20
5 in. distal	20	20	20	21½	20	20½
10 " "	28½	16	16	19¼	15¼	15½
15 " "	26½	13½	15¼	21½	13	15½
20 " "	25½	10¼	14½	20¼	11½	14½
25 " "	24	—	11	12¼	—	11

The measurements on Jan. 22, 1928, were taken in the evening, when at their worst ; they decreased with the night's rest.

COMMENTS.—The origin of the œdema remains obscure, but clearly the inflammatory attack in 1917 determined the onset of the elephantiasis. By operating on the limbs in sections, all shock or inflammatory reaction was avoided and sound healing obtained. The patient is now well into the fourth year since completion of treatment, and there has been no leaking or marked enlargement anywhere, so that, although some degree of recurrence is manifest, the condition remains satisfactory.

Case 3.—A man, age 24, admitted to the Hospital for Tropical Diseases on May 10, 1921, under Dr. G. Carmichael Low.

HISTORY.—Patient was born in Australia, and went to sea in 1909, trading chiefly between South Africa, Honolulu, and England ; served in Mesopotamia in 1916, when he contracted syphilis and gonorrhœa, which resulted in bilateral inguinal buboes together with fever, and swelling of the left leg. Both buboes were opened and the infected glands removed. Evidently the removal was radical, as primary healing followed. After this, occasional bouts of fever occurred, accompanied by swelling of scrotum and left leg. In 1919 in Australia he had three such attacks, and lymphangioplasty was done, carrying silk threads from the scrotum into the abdomen : the result was unsatisfactory. It was recorded then that no microfilariae were present in his blood. At Gibraltar in 1921 he suffered for six weeks from malarial fever, returning to England in March. When admitted to hospital benign tertian malaria parasites were present in his blood, but no microfilariae were found. The Wassermann reaction was fully positive. The scrotum was found to be moderately enlarged and thickened, as was the skin of the penis, especially on the dorsal aspect. Other systems were normal except for an enlarged spleen. While his syphilis and malaria were being treated the elephantoid tissue of the scrotum and penis was removed and plastic flaps formed, with good result.

COMMENTS.—As no microfilariae were ever found, as his chance of becoming infected with filariae was small, and as the beginning of the elephantiasis was definitely associated with his venereal infection and buboes, it seems reasonable to attribute the patient's condition to this source, a contributory factor being the too drastic clearing of the buboes at the operation. It is interesting to note the failure of the lymphangioplasty.

Case 4.—A man, age 47, admitted to the Hospital for Tropical Diseases on Feb. 3, 1922, under Dr. G. Carmichael Low.

HISTORY.—Born in India of white parents, patient came to England at the age of 5 years. From 1892 to 1903 he was a ship's steward serving in West Indian, Chinese, and Pacific waters ; then, in perfect health, obtained shore job. Married ; no venereal disease.

PRESENT ILLNESS.—In 1906, while gardening, he injured the knuckles of the left hand. Inflammation and swelling set in at once, with much pain. In three weeks the hand and forearm were greatly swollen, and the palm discharged at times.

The swelling gradually subsided, but use of the hand was lost. Later, attacks of inflammation occurred accompanied by increasing swelling, until by 1914 the fingers were as big as a normal wrist and the wrist was 24 in. in circumference. In 1914 venous thrombosis occurred first in the left leg, then in the right. In September, 1915, the arm was amputated above the elbow. Soon his feet and legs began to swell and were painful at times.

When admitted to hospital he was found to have elephantiasis of both legs, the toes being ulcerated and the skin pigmented. General and local palliative treatment was given, and the patient discharged slightly relieved.

COMMENTS.—While possibly of filarial origin, it appears likely that the elephantiasis was the outcome of the septic wound sustained in the left hand, and certainly to this can be attributed the venous thrombosis which paved the way for the elephantoid condition of the legs.

Case 5.—An Indian lascar, admitted to the Hospital for Tropical Diseases on June 17, 1921, suffering from elephantiasis of the scrotum and penis. *Microfilariae* were present in the patient's blood. The scrotum was rather smaller than a football, and was removed by Colonel J. J. Pratt without difficulty. The penis was left mostly denuded of skin. Three weeks later Thiersch grafts were applied with satisfactory results.

COMMENTS.—A straightforward example of filarial elephantiasis illustrating the need for skin-grafts.

Case 6.—We would cite again the case of elephantiasis that was reported by Dr. G. Carmichael Low.²⁰ Cancer causing elephantiasis must be so rare that no apology is made for a second reference to this patient, whose disability arose from cancer of the prostate.

SUMMARY.

1. Elephantiasis is a condition of hypertrophy and hyperplasia developing in a part as the result of excessive protein from lymphatic exudate.
2. This exudate is present as the result of infection, most often streptococcal, occurring in a part primarily presenting venous and lymphatic stasis.
3. Tropical elephantiasis has the same essential pathology as that occurring in temperate climates.
4. In treatment focal sources of infection must be sought and removed, and other pre-operative measures adopted.
5. Kondoléon's operation holds out good prospects of a prolonged temporary, if not permanent, cure.
6. Case reports are given illustrating various points in the pathology and treatment of elephantiasis.

Our best thanks are tendered to Dr. G. Carmichael Low for allowing us to cite patients admitted to his hospital beds, and for a critical survey of this paper: likewise to Dr. Manson-Bahr for helpful hints as to classification. Formerly Colonel J. J. Pratt suggested joint publication of Case 2, and we gratefully acknowledge his work and advice in connection with it, and with Case 5.

REFERENCES.

- ¹ KENNEDY, Abstr. by Busey, *New Orleans Med. and Surg. Jour.*, 1878, 529.
- ² CHEVALIER, *Med. Chir. Trans.*, London, 1817, ii, 63.
- ³ SOUTHAM, *Brit. Med. Jour.*, 1902, 1115.

- ⁴ MATAS, *Amer. Jour. Trop. Dis. and Prev. Med.*, 1913, i, 60.
- ⁵ UNNA, *Histopathology of Diseases of the Skin* (trans. Walker), 1896, 495.
- ⁶ HUTCHINSON, *Policlinic*, 1902, 289 and 291.
- ⁷ SABOURAUD, *Ann. de Dermatol. et de Syph.*, 1892, 552.
- ⁸ PROUT, *Brit. Med. Jour.*, 1908, 1364.
- ⁹ DUBREUILH, cited by Shattuck, *Boston Med. and Surg. Jour.*, 1910, 718.
- ¹⁰ ELLIOT, *Jour. of Cutan. and Gen.-urin. Dis.*, 1917, xxxv, 17; *Jour. Cutan. Dis. includ. Syph.*, 1917, xxv, 17.
- ¹¹ SISTRUNK, *Surg. Gynecol. and Obst.*, 1918, April, 388; *S. Amer. Med. Jour.*, 1921, Aug., 619; *Ann. of Surg.*, 1927, Feb., 190.
- ¹² BOCKHART, cited by Unna (*loc. cit.*).
- ¹³ ALBERCA, *Comptes rend. Soc. de Biol.*, 1924, 813.
- ¹⁴ WINTROP, *Jour. Amer. Med. Assoc.*, 1911, 1592.
- ¹⁵ MILROY, *N. Y. Med. Jour.*, 1892, lvi, 505.
- ¹⁶ HOPE and FRENCH, *Guy's Hosp. Rep.*, 1908, 5.
- ¹⁷ WHITE, *Brit. Med. Jour.*, 1924, i, 376.
- ¹⁸ SEQUEIRA, *Brit. Jour. Dermatol. and Syph.*, 1911, 57.
- ¹⁹ ADAMSON, *Ibid.*, 1910, 161.
- ²⁰ LOW, *Trans. Roy. Soc. Trop. Med. and Hyg.*, 1923, xvii, 77.
- ²¹ STARLING, *Principles of Human Physiology*, 1925, 1020.
- ²² *The Practice of Medicine in the Tropics*, ed. Byam and Archibald, 1923, iii, 1937.
- ²³ MITCHELL, *Brit. Med. Jour.*, 1909, ii, 1462.
- ²⁴ *London School of Trop. Med. Research Mem. Series*, v, Mem. 7.
- ²⁵ CARNOCHAN, *Erichsen's Science and Art of Surgery*, ii, 940.
- ²⁶ HANDLEY, *Brit. Jour. Surg.*, 1908, i, 783.
- ²⁷ LANZ, *Zentralb. f. Chir.*, 1911, xxxviii, 152.
- ²⁸ KONDOLÉON, *Ibid.*, 1912, xxxix, 1022; *Münch. med. Woch.*, 1912, lix, 2726.
- ²⁹ SAPPEY, *Anat. Phys. et Path. des Vaisseaux Lymphatiques*, 1874.
- ³⁰ HILL, *Surg. Gynecol. and Obst.*, 1915, xxi, 334.
- ³¹ ROYSTER, *Jour. Amer. Med. Assoc.*, 1914, lxii, 1720.
- ³² HENRY, *Brit. Jour. Surg.*, 1922, ix, 111.
- ³³ CHATTERJI, *Tropical Surgery and Surgical Pathology*, 1927, 113. New York: W. Wood & Co.
- ³⁴ BERTWISTLE, *Brit. Med. Jour.*, 1926, ii, 523.

THE DEPRESSIVE INFLUENCE OF THE SYMPATHETIC NERVES ON GASTRIC ACIDITY.

By H. MOLL,

MEDICAL TUTOR AND REGISTRAR IN THE UNIVERSITY OF LEEDS;

AND E. R. FLINT,

ASSISTANT SURGEON IN THE GENERAL INFIRMARY, LEEDS.

THIS study has been undertaken in order to obtain further information about the inhibitory influence, if any, of the sympathetic nerves on gastric acidity.

Evidence in favour of the depressive influences reaching the stomach by the splanchnic nerves will be deduced from:—

I. Clinical Observations: (1) Gastric analyses in cases of hyperthyroidism; (2) The effect of thyroid feeding on gastric secretion; (3) Changes produced by adrenalin and nicotine administration; (4) Depressive action of emotions on acid secretion.

II. Experimental Observations: Observations on HCl secretion after bilateral section of the splanchnic nerves in the dog.

I. CLINICAL OBSERVATIONS.

1. GASTRIC SECRETION IN GRAVES' DISEASE.

It has been a matter of some dispute whether in hyperthyroidism gastric secretion is raised or lowered. The mass of evidence, however, points to a tendency towards anacidity in this disease. Lockwood,¹ in a study of 90 cases of definite hyperthyroidism, made gastric analyses in 24 cases which presented digestive symptoms, and found that achlorhydria was present more often in severe cases, and that it was more frequent in cases of true exophthalmic goitre than in the *forme fruste* and in the adenomatous types. Of the 24 cases in which he made gastric analyses, 10 were achlorhydrias, 3 gave a hypochlorhydria, and 11 showed normal figures. He concluded that his findings suggested a tendency in hyperthyroidism towards a diminution rather than towards an increase of gastric secretion.

Barker² states that in exophthalmic goitre the gastric secretion as a rule is diminished but that there are certain cases in which it is increased. King³ reports increased stomach and intestinal motility with an achlorhydria in exophthalmic goitre. Wolpe⁴ found achylia constant in all cases of pronounced types of exophthalmic goitre, but the hydrochloric acid was unchanged in other types of hyperthyroidism. Leist⁵ found that of 8 cases of exophthalmic goitre 7 showed achlorhydria, and explained his findings on the basis of a subnormal protein content of the blood serum rather than of a hormonal action.

In contradistinction to these findings, others report hyperchlorhydria

in Graves' disease. Neilson⁶ makes the statement that hyperacidity is frequent in hyperthyroidism, and the same is reported by Boenheim.⁷ Maranon⁸ observed sometimes crises of hyperacidity. Sajous⁹ states that excessive thyroid secretion causes excessive metabolism in the gastro-intestinal mucous membrane and muscles, with increased secretion and motor activity, manifested by hyperchlorhydria, gastro-succorrhœa, frequent vomiting, diarrhœa, and serous light-coloured stools.

In view of these contradictory reports, largely due to scantiness of observations, one of us¹⁰ (H. M.) was led to make a more extensive study on 50 cases of hyperthyroidism. The cases were divided into five groups. The following table is a summary of the gastric analyses of these cases :—

Table I.—SUMMARY OF GASTRIC ANALYSES OF 50 CASES OF HYPERTHYROIDISM.

	ACHLOR- HYDRIA	HYPO- CHLOR- HYDRIA	NORMAL	HIGH N.
Chronic Graves' disease of more than 2 years' standing ..	15	6	1	1
Acute Graves' disease of less than 1 year's standing ..	4	4	3	—
Toxic adenoma	—	4	2	1
<i>Forme fruste</i>	3	—	2	1
Puberty hyperplasia	—	1	1	1
Total	22	15	9	4

The chief conclusions to be drawn from these findings are: (1) That there is a constant tendency towards subnormal or absent secretion of HCl in Graves' disease; (2) That the achlorhydria is more frequently found in cases of hyperthyroidism of long standing, while in the more acute cases the gastric secretion is least diminished; (3) That the secretion of HCl in cases of toxic adenoma and puberty hyperplasia is usually normal or subnormal but never absent.

These observations seem to offer an explanation why so many observers have made such contradictory statements, as it would appear that the achlorhydria in hyperthyroidism is an acquired condition and not constitutional in the patient, and that it occurs in the course of the disease, the cases of long standing presenting it in the most pronounced fashion. Furthermore, as the achlorhydria in Graves' disease has a maximal incidence in the more chronic cases, it is possible that it may be due to a persistent stimulation of the sympathetic system by the thyroid toxæmia.

2. EFFECT OF THYROID FEEDING ON GASTRIC ACIDITY.

The literature reveals even more contradictory reports on the effects produced on gastric secretion by experimental thyroid feeding in animals. Boenheim¹¹ states that after injections of thyroid extract the secretion of the stomach is raised. Rogers¹² and his associates found that in dogs with a Janeway fistula the subcutaneous injection of the watery saline extract of

the thyroid increased the gastric flow. The quantity of gastric juice and the total acidity were estimated for three 15-minute periods. It was concluded that the thyroid activates gastric secretion through the vagus by facilitating the absorption of 'food' by the cells and the metamorphosis of this food into the cell's secretion.

Truesdale¹³ made use of a Pawlow pouch and gave 0.3 gm. to 1 gm. of thyroid extract per kilo. of body weight, and observed in all cases a decrease in the continuous secretion of gastric juice. In discussing the results he offers the following explanations: The diminished secretion might be a secondary result of disturbed metabolism and other abnormal conditions which might produce changes in the blood constituents, thus affecting the activity of the secreting cells; and lastly the thyroid might act through a disturbance of the nervous mechanism of gastric secretion by inhibiting the nervous impulses of appetite or by stimulation of inhibitory secretory nerves or by inhibition of secretory nerves.

Hardt¹⁴ made two experiments on dogs, weighing 2.5 and 4 kilo. respectively, which were fed with 10 gm. of Armour's desiccated thyroid daily over a period of two weeks. The collection of gastric juice was begun one hour before feeding and continued for one hour after feeding. The controls were made by feeding the same quantity of meal in the absence of the thyroid. As a result of these experiments Hardt found no indication of either hyperacidity or hypersecretion following thyroid administration. In both dogs, on the contrary, there was a tendency toward depression of the acidity and of the rate of secretion. The dogs were in perfect health, and to all appearances the thyroid had no toxic effects. The acidity and the volume of the gastric juice returned to normal a few days after the thyroid feeding was discontinued.

It is difficult to gauge to what extent thyroid feeding influences gastric secretion from the observations mentioned above, as the gastric analyses in most cases have been carried out over too short a period—three-quarters of an hour in the experiments of Rogers and his associates, and one hour in those made by Hardt. Moreover, only the volume of gastric secretion and the total acidity were estimated, not the free HCl. We have therefore thought it well to repeat these experiments on four dogs with a Janeway fistula, fractional gastric analyses being carried out over periods of several hours. Thyroid extract (Burroughs & Wellcome 'tabloid' thyroid gland) was administered to the dogs in increasing doses until a definite loss of weight was obtained, amounting to 5 to 10 per cent of the body weight of the animal. The amount of thyroid extract required to produce this was found to be equivalent to 1 gm. of fresh gland per kilo. of body weight of the animal. Fractional gastric analyses conducted under exactly the same conditions were made before and after thyroid feeding and the results compared. The test-meal was the standard oatmeal gruel, of which a pint was given to the dog; sometimes the animal would refuse to take the full amount, but always took sufficient for the gastric analysis to be made. The total acidity and the free HCl were estimated over a period of five hours. As it was found that free HCl is not secreted after an oatmeal gruel test for at least an hour, an interval of an hour was allowed between the feeding time and the taking

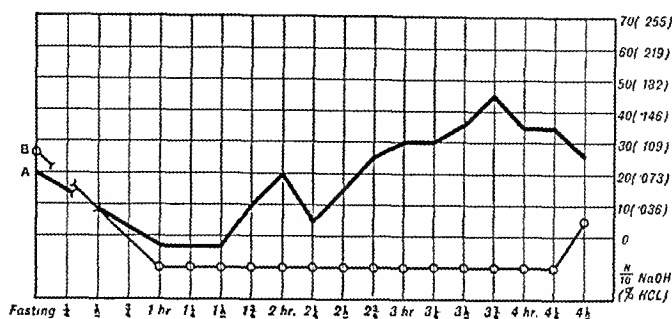


FIG. 247.—Thyroid feeding. Dog V (see Protocol 2).
A, Free HCl before feeding; B, After 8 days of feeding.

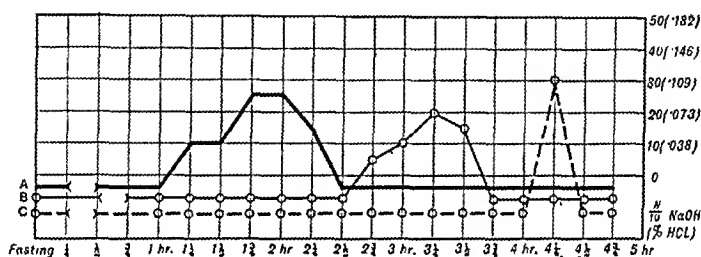


FIG. 248.—Thyroid feeding. Dog VII (see Protocol 1).
A, Free HCl before feeding; B, After 12 days of feeding; C, After 21 days.

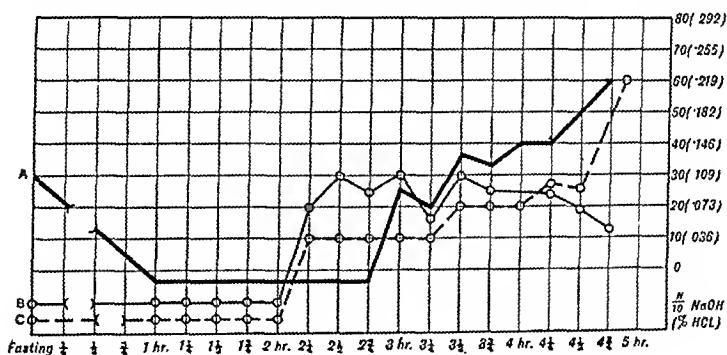


FIG. 249.—Thyroid feeding. Dog III (see Protocol 3).
A, Free HCl before feeding; B, After 8 days of feeding; C, After 15 days.

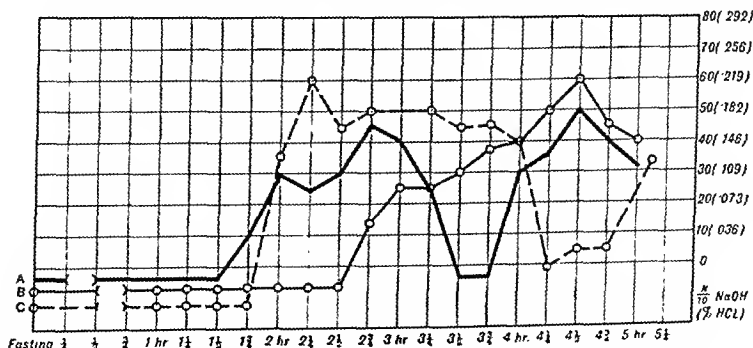


FIG. 250.—Thyroid feeding. Dog VI (see Protocol 4).
A, Free HCl before feeding; B, After 12 days of feeding; C, After 21 days.

of the first sample. The dogs were in a good state of health throughout the whole time of thyroid feeding, but towards the end of the course became rather restless and irritable. The urine was frequently tested for sugar, but none at any time developed glycosuria. At the end of this paper protocols of the experiments are given. In two cases (*Dogs V and VII*) thyroid feeding produced almost complete achlorhydria, free HCl being absent in all the samples except in one towards the end of the examination. In these two cases, therefore, thyroid-gland feeding markedly delayed and inhibited free HCl secretion (*Figs. 247, 248*). One of these dogs, however, died during the course of thyroid feeding, and it is possible that the state of bodily depression preceeding death may partly account for the lowering of gastric secretion in this case.

In the third dog (*Dog III*) the curve of free HCl was only very slightly lowered, if at all (*Fig. 249*), while in the fourth experiment (*Dog VI*) thyroid feeding produced a slight rise in acidity (*Fig. 250*).

It is therefore difficult to draw any conclusions from these results. Thyroid administration certainly does not lead to lowering of gastric secretion in all cases, while in some it may even raise it. These experiments would seem to explain why different observers have reported contradictory results.

3. CHANGES PRODUCED BY ADRENALIN AND NICOTINE.

Adrenalin.—It is interesting to note the effect which adrenalin has on gastric secretion, because, according to Langley,¹⁵ adrenalin and certain related substances produce effects which are, in nearly all cases, like those brought about by stimulating the sympathetic nerves; although there are exceptions, such as the case of the sweat glands, where sympathetic stimulation has a marked effect and adrenalin has none.

There is a great mass of evidence, gathered from experiments by Elliott,¹⁶ Tumpowski,¹⁷ and more recently by Brown and McSwiney,¹⁸ that adrenalin inhibits the motor activity of the stomach, and a similar action has been observed on the secretory function of the stomach. Rogers,¹² together with others, found that on dogs with a Janeway fistula injection of epinephrin or of suprarenal nucleoproteins inhibited gastric secretion. Lim¹⁹ states that adrenalin does not inhibit the secretion, but that it may delay its outflow from the stomach. His results, however, are opposed to those of Hess and Gundlach.²⁰ These authors found a strong inhibitory action of adrenalin on gastric secretion in all their experiments, the intensity of the effect depending on the amount of adrenalin administered. The inhibitory action was found to be greater on the volume of gastric juice than on the total acidity and free HCl. One of us (H. M.) tested the action of adrenalin on two cases of hyperchlorhydria in man, and the results proved that adrenalin has, on the contrary, a definite inhibitory action also on the secretion of free HCl. The first experiment was carried out on a case of hyperchlorhydric dyspepsia without clinical or radiological evidence of gastric or duodenal ulceration. A test-meal followed by a fractional gastric analysis showed a hyperchlorhydria with a climbing type of curve. A second test was made ten days afterwards, and at the end of the first hour a subcutaneous injection of 15

minims of 1-1000 adrenalin was given. The charts showed a surprising change (Figs. 251, 252). Following the injection of adrenalin the HCl curve dropped down to zero, only to rise again to even a higher level within the second hour.

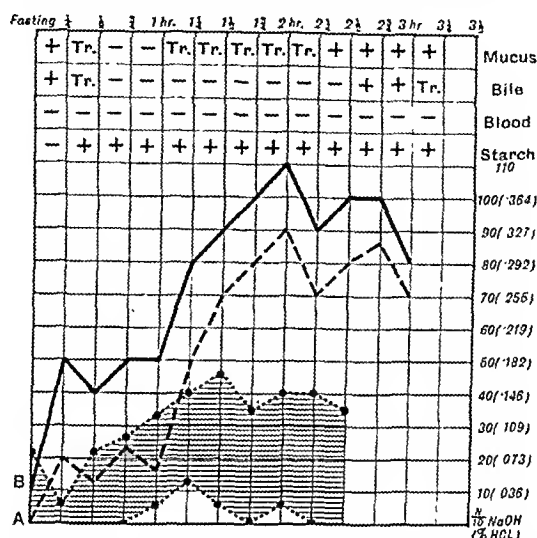


FIG. 251.—Before the injection of 15 min. of 1-1000 of adrenalin.
A, Free HCl; B, Total acidity.

[The shaded area in Figs. 251-256 represents the limits for free HCl of 80 per cent of normal people.]

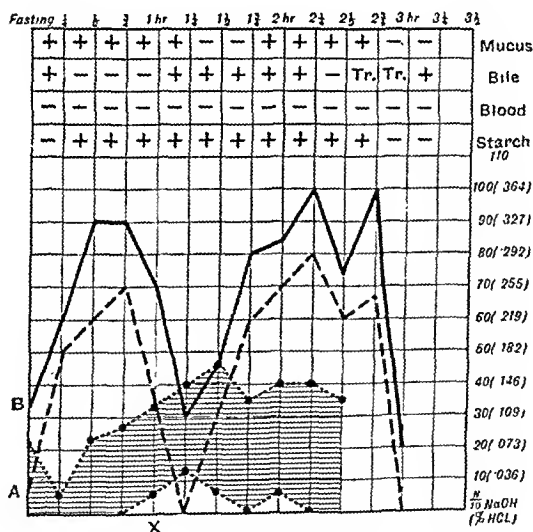


FIG. 252.—Same case as Fig. 251 after injection of adrenalin at point marked X.

Impressed by this, but in doubt whether the sympathetic stimulation was due to adrenalin or the prick of the needle, we decided to treat a similar case with adrenalin over some time. To another case of hyperchlorhydria with a high and climbing curve was administered 5 minims of 1-1000 adrenalin subcutaneously every four hours over a period of two and

a half days, until after the fifteenth injection there occurred abruptly symptoms of sympathetic stimulation, viz., tachycardia, palpitation, and tremor. Thereupon a second fractional gastric analysis was made. This showed a striking lowering of the HCl curve, though the emptying time of the stomach

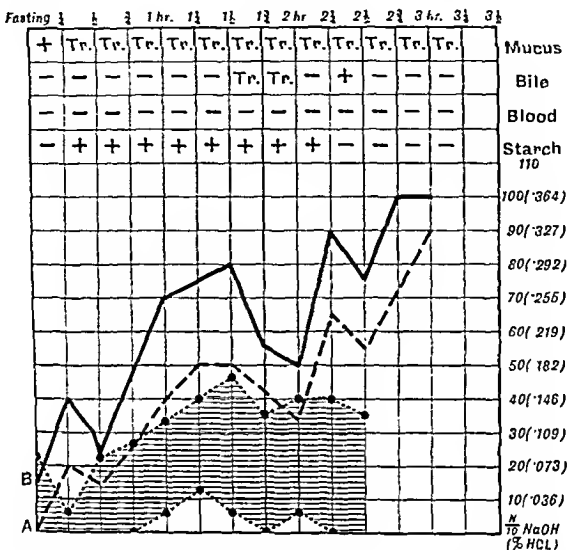


FIG. 253.—Before giving 15 injections of 5 min. of 1-1000 adrenalin at four-hourly intervals over a period of two and a half days. A, Free HCl; B, Total acidity,

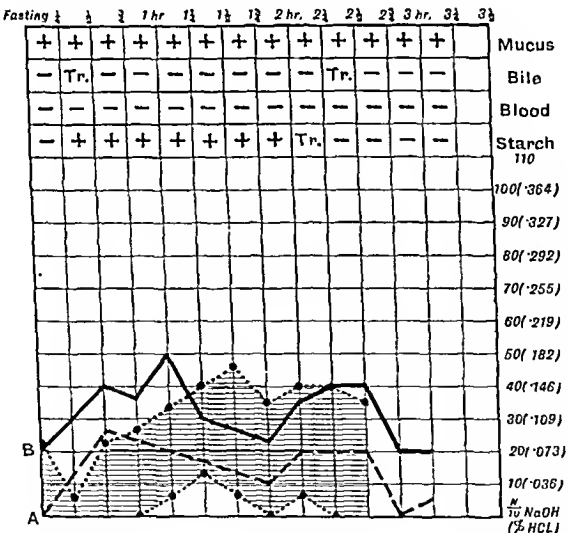


FIG. 254.—Same case as Fig. 253 after the injections of adrenalin.

and the amount of duodenal regurgitation had remained the same. It is interesting to record that, following the injections, the patient felt considerably better and was free from pain for several days. (Figs. 253, 254.)

Nicotine.—If stimulation of the sympathetic causes this change in acid

secretion, then paralysis of the sympathetic should have the opposite effect. The drug best known to have such an action is nicotine, inasmuch as Langley has shown that it paralyzes the preganglionic sympathetic cells. It has already been noted by many observers how smoking increases the secretion of HCl

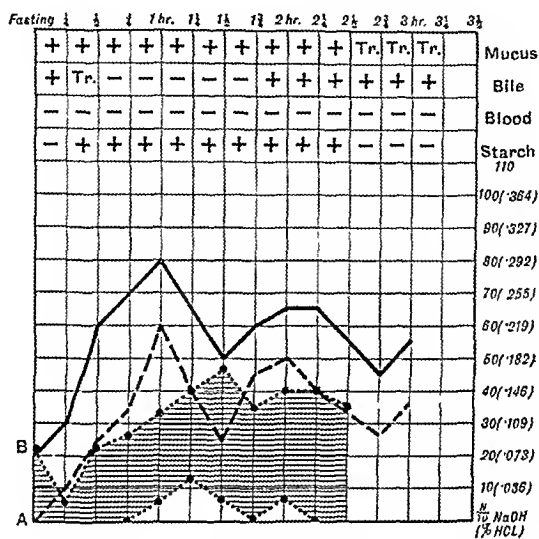


FIG. 255.—Before giving 30 injections of $\frac{1}{2}$ gr. of nicotine thrice daily over a period of ten days. A, Free HCl; B, Total acidity.

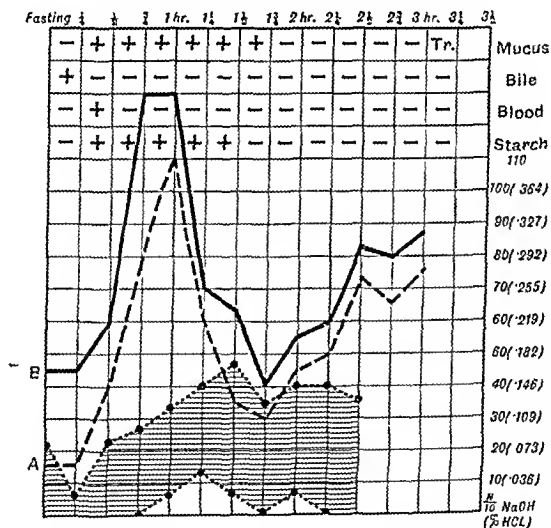


FIG. 256.—Same case as Fig. 255 after the injections of nicotine.

and tends to produce hyperchlorhydria. In a not very extensive review of the subject we failed to find in the literature any report on the action of nicotine on gastric secretion when given in the form of the pure alkaloid by hypodermic injection. The results given below may, therefore, be of some

interest. One of us (H. M.) had the opportunity of observing the action of nicotine on gastric acidity when this drug was administered to a series of cases of post-encephalitic Parkinsonism in an attempt to reduce plastic tone.²¹ Nicotine, in the form of pure alkaloid, was administered hypodermically thrice daily in doses varying from $\frac{1}{10}$ to $\frac{1}{8}$ gr. over a period of two to three weeks. In six of these cases fractional gastric analyses were made before and after the course of treatment. One case shows no change in gastric acidity. The other five all show an increase in HCl secretion. In one case (Figs. 255, 256) the curve rose from a normal chlorhydria to a very definite hyperchlorhydria after ten days of nicotine treatment, $\frac{1}{8}$ gr. of the drug having been administered thrice daily (30 doses of $\frac{1}{8}$ gr., or a total dose of 6 gr.).

If deductions may be drawn from these experiments, one is justified in concluding that adrenalin lowers gastric acidity by stimulation of the sympathetic, whereas nicotine raises it by paralyzing the sympathetic.

4. THE DEPRESSIVE ACTION OF EMOTIONS ON GASTRIC ACIDITY.

Cannon²² found that in a case of any sign of rage or distress, or mere anxiety, was accompanied by a total cessation of the movements of the stomach, and that even slight psychic disturbances were accompanied by stoppage of peristalsis. His observations were confirmed by Auer²³ and by Lommel. Furthermore, it would appear from other experiments that the sympathetic system for the greater part, and the vagus to a less extent, provide the course for these inhibitory impulses. Thus, lasting inactivity of the gastro-intestinal tract was produced reflexly by trauma of the testicles in certain experiments made by Murphy and Cannon²⁴ on the dog, while these observers noted that, if the splanchnic nerves had been severed previously, trauma of the testicles had no effect whatever. Respiratory distress was also shown to inhibit gastric motility reflexly only when the sympathetic and the vagus nerves were intact; when these were cut, even a marked degree of respiratory distress failed to stop the movements of the alimentary canal. As counterpart to this inhibitory action of emotions on gastric motility, interesting observations—although unfortunately few in number—have been made on a similar depressive influence on the secretory function of the stomach by unpleasant emotions. According to Langdon Brown²⁵ the antagonistic action of the sympathetic is more clearly demonstrated in the inhibition of salivary secretion than of gastric or pancreatic. This inhibitory action is well seen in the dry mouth of fear, which is the physiological basis for the old Indian 'rice ordeal' in which persons suspected of crime were given consecrated rice to chew. The man who spat it out dry was adjudged guilty, for the fear of detection had stopped salivary secretion. But there can be little doubt that the sympathetic has a corresponding inhibitory action on gastric secretion. Bennett and Venables²⁶ have observed the variations of gastric acidity in man during hypnosis, and by this very ingenious method of evoking emotions found that suggestion of nausea caused delay of emptying and inhibition of gastric secretion, while suggestion of hunger on the contrary caused rapid emptying and a rise of acidity. Great anxiety (such as produced by suggesting to the

hypnotized subject, an old army man of the Flying Corps, that he was involved in an aeroplane smash) caused strong inhibition with delayed emptying rate.

Some interesting observations have also been made on the effect of fright on gastric secretion in dogs by Bickel and Sasaki.²⁷ These authors report that, by frightening dogs during the process of conducting a fractional analysis of the gastric juice, they were able to note a considerable fall in the volume, though the acidity was not much influenced.

II. EXPERIMENTAL OBSERVATIONS.

THE EFFECTS ON HCl SECRETION OF SECTION OF THE SYMPATHETIC NERVOUS SUPPLY OF THE STOMACH.

Few experiments have been made with a view to ascertaining the changes in acid secretion of the stomach after section of its sympathetic nervous supply. Schupfer²⁸ excised simultaneously and bilaterally the anterior and posterior nerve-roots lying between the 4th and 8th or between the 5th and 9th dorsal segments in the dog, and found as a constant result an increase in gastric acidity. This rise of acidity depended for the most part on an increase of combined HCl, and to a less extent on that of free HCl. Gaultier²⁹ found that stimulation of the splanchnics or of the solar plexus did not affect gastric acidity, whereas section of the splanchnics and avulsion of the solar plexus gave rise to a constant and pronounced hyperchlorhydria.

Apart from these two observations, from a not very complete review of the literature, we failed to find any more reports of similar experiments. The following is an experimental study which was undertaken to ascertain the effects of bilateral section of the splanchnics on free HCl secretion.

EXPERIMENTAL TECHNIQUE.

In order that the results obtained in experimental conditions in animals may be assessed at their true clinical value, it is essential that the experimental observations should be carried out under similar conditions to those which are in use in the clinical laboratory. We have therefore endeavoured to carry out fractional gastric analyses in the dog on similar lines to those of an ordinary standard Rehfuess test-meal. The only difference between the former and the latter was the way in which the tube was introduced into the stomach. As it was impossible to introduce the Rehfuess tube through the mouth in the dog, we adopted a Janeway fistula, this being the least likely to interfere with the nervous supply of the stomach; it was made in the following way. General anaesthesia was induced and maintained by the open method, ether being used throughout. A paramedial incision of the abdomen was employed, and before proceeding to make the fistula the left splanchnic nerve was exposed and cut; this was done because otherwise the approach to the left splanchnic nerve would have been made very difficult by the presence of the fistula. Ducceschi³⁰ has brought evidence to show that afferent fibres in each splanchnic nerve are connected through filaments from the coeliac plexus with the entire surface of the stomach. The section of only one splanchnic would therefore not interfere with the nervous supply of the

stomach. McCrea, McSwiney, and Stopford³¹ have lately proved that this is also true for the vagus, as the motor activity of the stomach is unaffected by unilateral vagotomy.

Under an aseptic technique a clamp was applied to the anterior surface of the stomach close to and parallel with the greater curvature and as near as possible to the cardiac end. The clamped piece, being an oblong about $2\frac{1}{2}$ in. long, was cut so as to leave it attached by rather a broad base at the cardiac end of the clamp. The main section on the stomach and the partially detached piece were closed by a continuous musculo-mucous suture, and over that a continuous peritoneal stitch. The final result was a suture line just anterior to the greater curvature of the stomach, and leading off from the cardiac end of this a peritoneal covered tube continuous with the cavity of the stomach and having a lumen about the size of a No. 10 rubber catheter. This tube was brought through a stab wound in the left rectus and sutured to the skin. A short piece of a Rehuss tube was inserted along this diverticulum into the main body of the stomach and fixed in position by a stitch. A Jancway fistula provides a very efficient method for easily introducing a Rehuss tube into the stomach, while it does not leak between examinations. The only difficulty experienced with this method was to keep the stoma open for any length of time once the tube came loose, unless it was passed subsequently every day.

After the normal secretion had been ascertained by repeated examinations, the animal was again placed under general ether anaesthesia and the right splanchnic nerve excised. Fractional gastric analyses were then again resumed at intervals varying from one week to several months after the operation.

The dogs were starved overnight or for at least twelve hours prior to taking a fractional gastric analysis. The fasting contents of the stomach were completely withdrawn, and the standard test-meal of one pint of oatmeal gruel, as employed by most workers on fractional gastric analyses in man, was then given. The dogs were trained to take the meal without being forced, by gradually accustoming them to it some time before beginning the experiments; most of the dogs, however, took to the oatmeal gruel without any difficulty and would lap it up heartily. Quarter-hour specimens of 2 c.c. of stomach contents were drawn off, after the lapse of an hour from the feeding time, as it was found that practically no free HCl is secreted during the first hour in the dog's stomach after an oatmeal gruel test-meal. The fractional observations were continued for $4\frac{1}{2}$ to $5\frac{1}{2}$ hours. The free HCl was estimated by titration with standard alkali to the turning-point of Toepfer's reagent; the total acidity by treating the juice with standard alkali until a pink colour with phenolphthalein resulted. In some of the earlier fractional analyses the total chlorides were also determined by the direct application of the Volhard Harvey method, in which an excess of standard silver nitrate solution together with an acidified iron-alum indicator is added to the juice and the excess of silver nitrate titrated with standard thiocyanate solution.

From these results a curve was plotted out representing fractional analyses of stomach juice over periods of $4\frac{1}{2}$ to $5\frac{1}{2}$ hours after the taking of the meal.

A post-mortem examination was made on every animal, and the section of the splanchnic verified both at autopsy and by histological examination of the excised piece of nerve.

EXPERIMENTAL FINDINGS.

Fourteen dogs have been operated on and over 50 fractional gastric analyses have been made. Reports are, however, given here only on 9 dogs and on 31 gastric tests, the remainder having been unsatisfactory. Of these 9 dogs, observations were successful after bilateral section of the splanchnic nerves in 4, while the other 5 dogs, though unsatisfactory in this respect, enabled us to ascertain the normal range of gastric secretion during a fractional test.

For the sake of brevity we have collected our results into tables. *Table II* summarizes all our observations with regard to certain aspects of gastric secretion, both before and after sympathetic denervation of the stomach. From this table we have worked out *Table III*, which contains the percentage

Table II.—SUMMARIES OF OBSERVATIONS ON NINE DOGS FOR GASTRIC SECRETION

EXPERIMENTAL ANIMALS	TOTAL NO. OF OBSERVA- TIONS MADE ON EACH ANIMAL.	NO. OF OBSERVA- TIONS IN WHICH BILE WAS PRESENT IN FASTING JUICE	NO. OF OBSERVA- TIONS IN WHICH FREE HCL WAS PRESENT IN FASTING JUICE	RANGE OF VALUES OF FREE HCL IN FASTING JUICE	RANGE AND MEAN VALUE OF TOTAL ACIDITY FASTING JUICE	
					Range	Mean Val.
(Controls)—						
Dog I	4	2	0	0	10-115	40
Dog II	1	1	0	0	—	—
Dog IV	2	1	1	0-55	45-75	60
Dog V	2	2	2	10-20	60-75	65
Dog IX	1	0	0	0	?	?
(Before Splanchnic Division)—						
Dog III	3	3	1	0-30	25-60	30
Dog VI	1	1	0	0	—	30
Dog VII	1	1	0	0	—	20
Dog VIII	1	1	0	0	—	10
(After Bilateral Section of Splanchnic Nerves)—						
Dog III	4	2	3	0-50	15-75	45
Dog VI	3	3	2	0-55	15-80	40
Dog VII	3	2	1	0-25	15-50	25
Dog VIII	5	0	3	0-75	15-90	35

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incidence of normal findings on all dogs, including those (*III, VI, VII, VIII*) which afterwards had a bilateral sympathetic division. *Table IV* has been plotted to show the contrast between the findings before and after splanchnic section and is confined, therefore, entirely to observations made on *Dogs III, VI, VII, and VIII*. Protocols of the experiments are given at the end of the paper.

From *Tables II and III* some generalities may be drawn as to the normal features of gastric secretion after a standard test-meal in the dog.

Rate of Emptying.—This was remarkably constant. The stomach was never found to be empty at the end of the test even when fractional observations were carried out for 5½ hours. This is rather in contrast with the findings of Cannon,²² who says that the stomach is usually empty about 3 hours after feeding the standard amount of carbohydrates. It is, however, in accord with the observations of McCrea, M'Swiney, and Stopford,³¹ who noted an average total emptying time ranging from 5 to 7 hours. The first observer used a mixture of 25 c.c. of starchy paste, boiled rice or boiled potatoes, with

BEFORE AND AFTER SYMPATHETIC DENERVATION OF THE STOMACH.

NO. OF OBSERVATIONS IN WHICH DUODENAL REFLUX WAS :			NO. OF OBSERVATIONS IN WHICH THE RISE OF ACID CURVE (FREE HCL) OCCURRED DURING THE :			MEAN AND MAXIMAL VALUES OF FREE HCL		MEAN AND MAXIMAL VALUES OF TOTAL ACIDITY		RATE OF EMPTYING : STOMACH NOT EMPTY AT THE END OF TEST (NO. OF OBSERVATIONS)
Absent	Slight	Marked	2nd Hour	3rd Hour	4th Hour	Mean	Maximal Value	Mean	Maximal Value	
2	2	0	1	3	0	—	75	—	120	4
0	1	0	1	0	0	—	35	—	80	1
0	2	0	0	1	1	40	45	85	100	2
0	2	0	2	0	0	40	45	90	115	2
1	0	0	0	1	0	—	60	—	90	1
3	0	0	0	2	1	60	110	115	160	3
1	0	0	1	0	0	—	45	—	110	1
0	0	1	1	0	0	—	25	—	110	1
1	0	0	0	1	0	—	70	—	110	1
3	1	0	4	0	0	80	95	100	125	4
0	0	3	3	0	0	60	70	65	95	3
2	1	0	2	1	0	25	30	55	60	3
1	1	0	2	0	3	75	95	100	125	5

5 grm. of bismuth subnitrate; the last instead used a mixture of 200 c.c. of milk, 60 grm. of bread, 7 grm. of bismuth sulphate, and 1 grm. of sugar. Allowances have also to be made for the difference in the two methods of estimating the emptying time, as it is common experience in clinical work to find marked discrepancies between the emptying rate as gauged by X rays and that obtained by the examination for the presence of starch in a fractional gastric test.

Table III.—FINDINGS IN NORMAL DOGS.

	PER CENT
Bile present in fasting juice	75
Free HCl present in fasting juice	25
Duodenal reflux, none	50
" " slight	43
" " marked	7
Rise of acid curve (free HCl), within 2 hours	37
" " " " " 3	50
" " " " " 4	13
Rate of emptying: stomach not empty at the end of the test	100

Fasting Juice.—We have not taken note in our observations of the volume of fasting juice, as little information can be drawn from it owing to the difficulty of ensuring the total withdrawal of the fluid. Free HCl was present in the fasting juice only in 25 per cent, while bile was present in 75 per cent of cases. From this it would seem that achylia, though not a constant finding, is a common occurrence in the fasting juice of the dog, and that the absence of free HCl in the fasting juice may be due either to lack of its continuous secretion during fasting time or to duodenal regurgitation. Other observers have also noted this absence of free HCl from the continuous secretion in the dog with a Pawlow fistula (Carlson), although some have denied it (Bickel and Rheinboldt³²).

Type of Curve.—On repeating gastric analyses on the same dog under identical conditions at frequent intervals, we found that although wide variations do often occur in the type of curve, the dissimilarity is often more apparent than real. Thus the acid curve tends to rise at a constant time and reaches a peak of maximal value which is also more or less constant. The same has been observed by Bell and MacAdam³³ in man. Before splanchnic division the acid curve tends to rise within the 2nd or 3rd hour, although quite frequently it may be delayed until the 4th or even 5th hour. There is little tendency towards duodenal regurgitation, and this was borne out both by the frequent absence of bile throughout the test meals, and also by the estimation of total chlorides, which gave a curve following very closely that of the free HCl.

Results after Splanchnic Division.—The following observations have been made on four dogs which survived the bilateral section of the major splanchnic nerves. Some of the results have been collected in statistical form in *Table IV*, although we have made use of the graph method to denote the changes in gastric acidity, as we do not consider that statistical methods can be reasonably applied to the elucidation of such curves.

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Continuous acid secretion is increased after bilateral splanchnic division, as shown by the greater frequency with which free HCl is found in the fasting juice, the higher values attained, and the comparative absence of bile (*Table IV*). There is furthermore a more prompt response of acid secretion to the

Table IV.—FINDINGS IN EXPERIMENTAL DOGS BEFORE AND AFTER OPERATION.

	PER CENT	
	Before	After
Bile present in fasting juice	100	47
Free HCl present in fasting juice	17	60
Duodenal reflux, none	84	60
" " slight	0	20
" " marked	16	20
Rise " of acid curve (free HCl), within 2 hours	34	74
" " " " " 3 " "	50	6
" " " " " 4 " "	16	20
Rate of emptying: stomach not empty at the end of the test	100	100

meal, the acid curve rising within the 2nd hour in 74 per cent as opposed to 34 per cent before the sympathetic denervation. Duodenal regurgitation does not seem to be appreciably influenced, although it is slightly more frequent after than before splanchnic division. The values of free HCl were definitely raised in three out of four experiments, as the charts (*Figs. 257-260*) show. The continuous line represents the acid curve of free HCl before

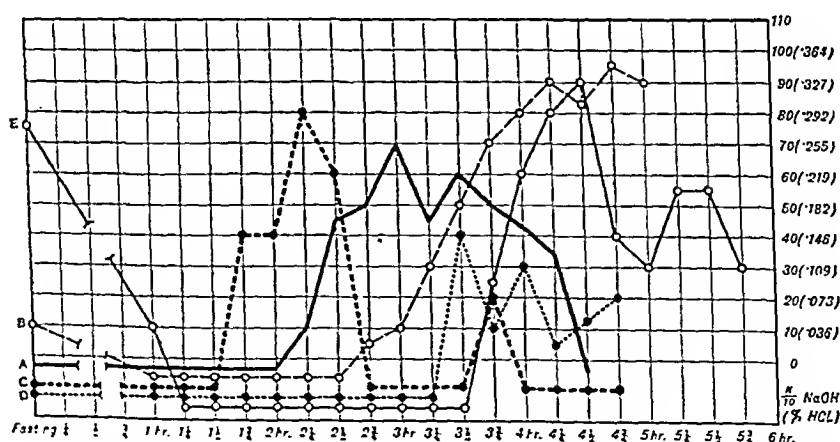


FIG. 257.—Bilateral section of the splanchnic nerves. Dog VIII (see Protocol 8). A, Free HCl before denervation; B, 13 days after; C, 22 days after; D, 32 days after; E, 7 months after.

splanchnic division, while the dotted lines represent the findings on different occasions and at varying intervals after sympathetic denervation of the stomach. The hyperchlorhydria induced by the secretion of the splanchnics

is rendered more apparent when one compares the acid curve before splanchnic division with the highest curve after denervation.

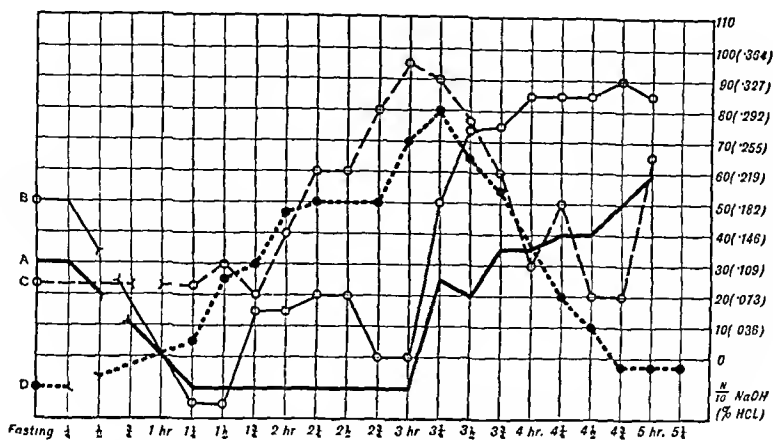


FIG. 258.—Splanchnic division. Dog III (see Protocol 5).
A, Free HCl before denervation; D, 7 days after; C, 3 weeks after; B, 3½ months after.

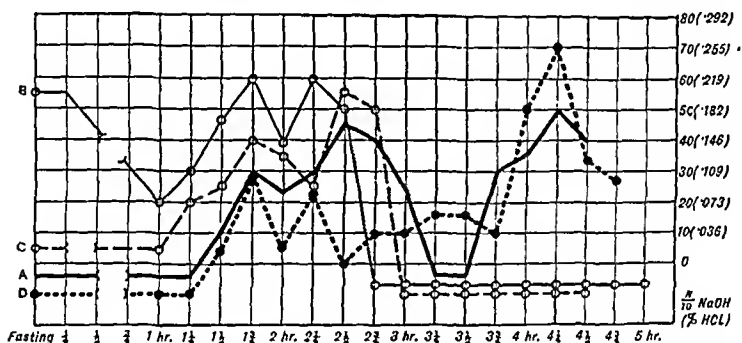


FIG. 259.—Splanchnic division. Dog VI (see Protocol 6).
A, Free HCl before denervation; D, 6 days after; B, 13 days after; C, 30 days after.

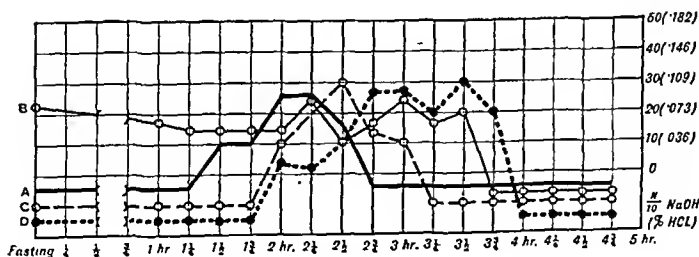


FIG. 260.—Splanchnic division. Dog VII (see Protocol 7).
A, Free HCl before denervation; B, 12 days after; D, 22 days after; C, 28 days after.

No mention is made of the variations of the total acidity values, as these do not seem to afford any information as to the true state of acid

secretion of the stomach. They were, however, found to follow very closely those of the free HCl (*Figs. 261-268*).

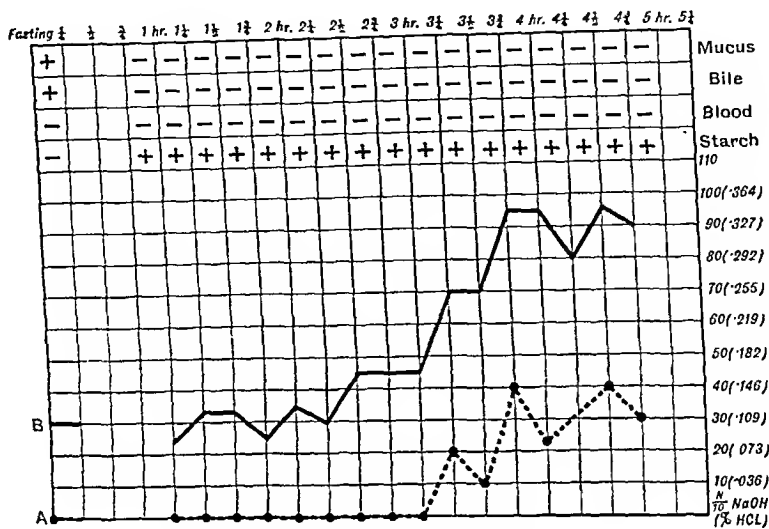


FIG. 261.—Dog III before splanchnic section.
A, Free HCl; B, Total acidity.

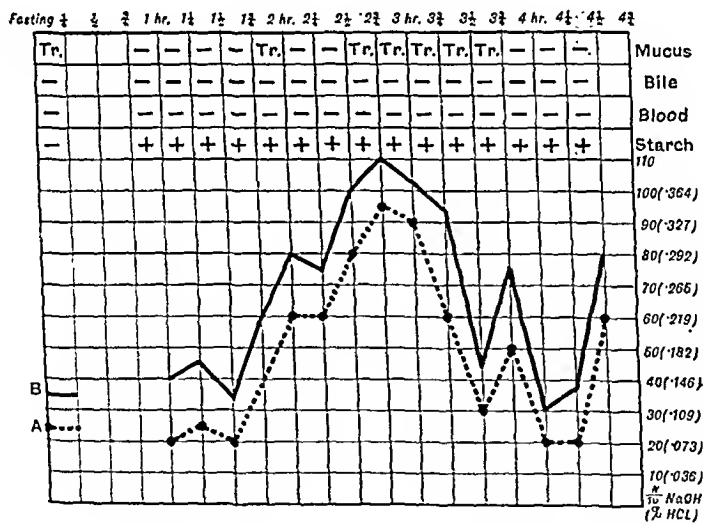


FIG. 262.—Dog III one month after splanchnic section.

DISCUSSION.

The interpretation of the results obtained from these experiments is subject to certain reservations and to criticism.

In the first place it is difficult to ascertain whether the division of the major splanchnic nerves ensures a complete sympathetic denervation of the stomach. It is quite likely that it does not, as sympathetic fibres may reach the stomach through the outer coats of blood-vessels, and furthermore the

peripheral intrinsic sympathetic nervous system of the stomach may compensate any loss of control due to central denervation. In this respect the stomach may be compared to the heart; like the heart it is essentially an automatic organ; its stimuli to movement and secretion arise within itself;

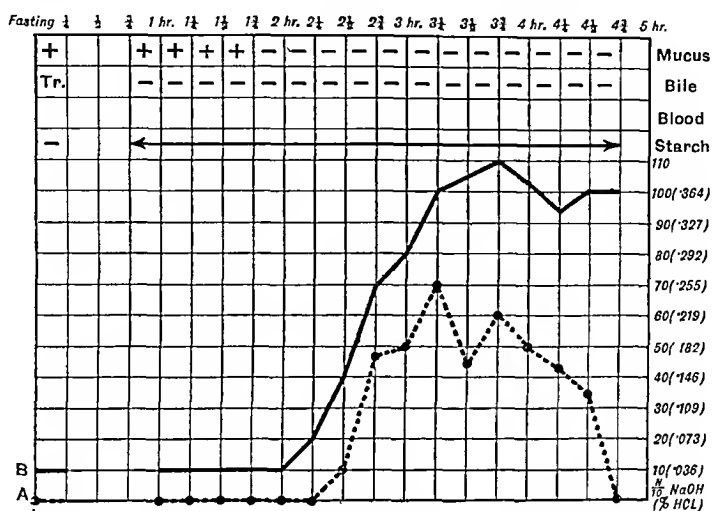


FIG. 263.—Dog VIII before splanchnic division.
A, Free HCl; B, Total acidity.

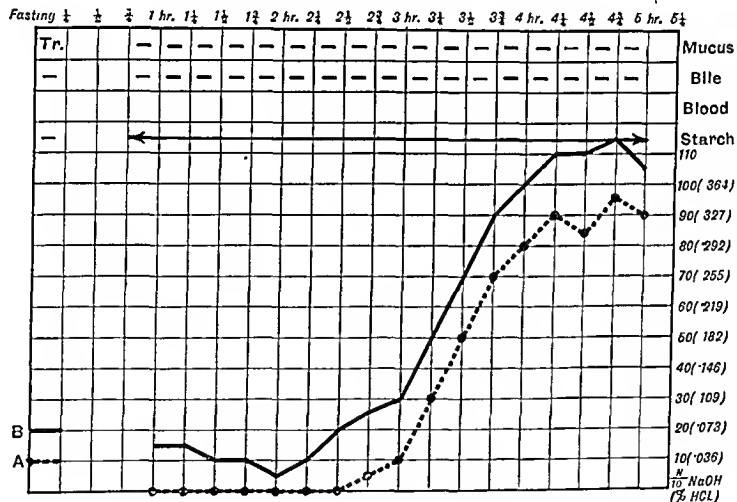


FIG. 264.—Dog VIII two weeks after splanchnic division.

but these movements and secretions are also regulated by the action of the extrinsic nerves, so as to adapt them to varying conditions.¹ It is highly probable that inhibitory fibres of gastric secretion are contained in the vagi, and this may overbalance any effect which division of the splanchnic nerves might have. It is therefore not surprising to find some inconsistency in the

results after splanchnic division, and that the hyperchlorhydria observed is only a relative one and not absolute.

In the second place the fractional gastric analyses after a test-meal rarely afford reliable information as to whether the stomach is able to secrete or

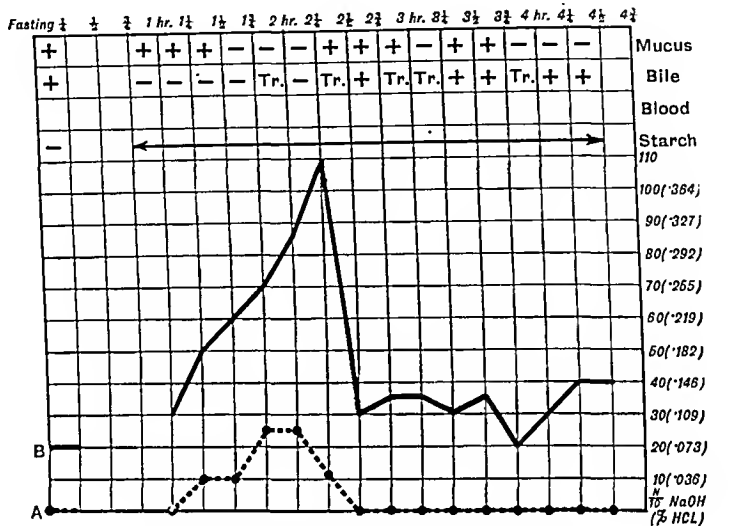


FIG. 265.—Dog VII before splanchnic section.
A, Free HCl; B, Total acidity.

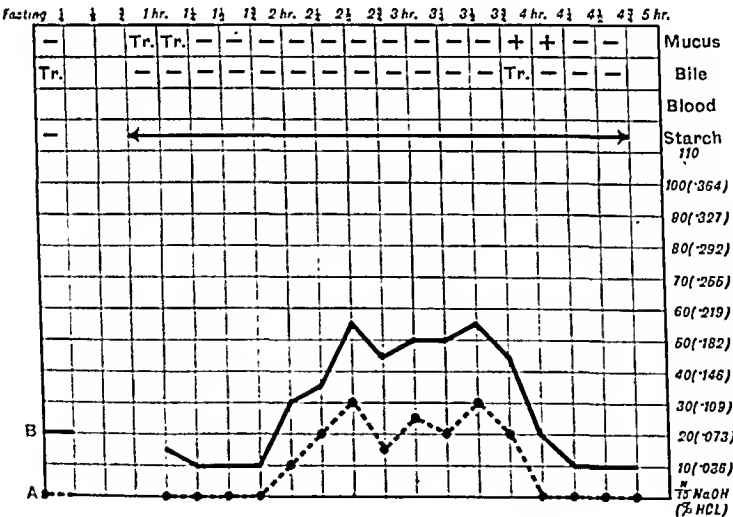


FIG. 266.—Dog VII three weeks after splanchnic section.

not, and as to the real acidity of the stomach contents. The admixture of food with gastric juice obscures the volume of the response, while the real acidity is masked by the neutralizing and diluting power of the food mass, evacuation into the duodenum, regurgitation, and retention.

Lastly, considerable variations in the acid values occur both before and

after sympathetic denervation, when taken on different occasions, and render comparisons very difficult. Although more accurate results are obtained by other methods of gastric analysis, such as the double intubation technique employing continuous aspiration (Lim, Matheson, and Schlapp³⁴, McBaird,

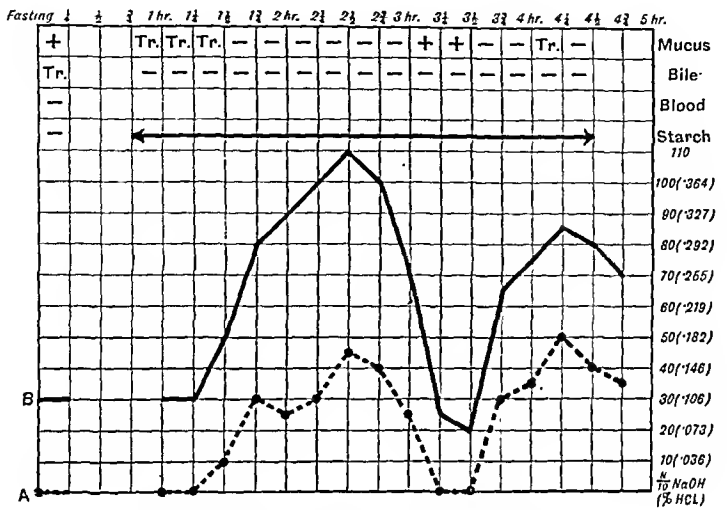


FIG. 267.—Dog VI before splanchnic division.
A, Free HCl; B, Total acidity.

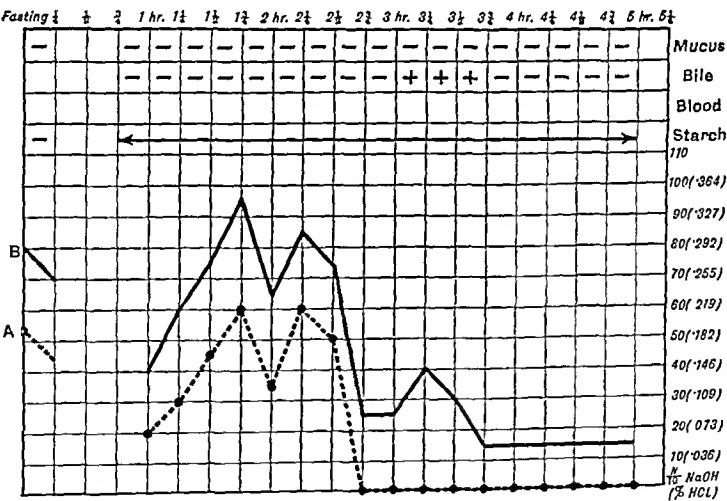


FIG. 268.—Dog VI two weeks after splanchnic division.

Campbell, and Hern,³⁵ or those of Heidenhain and Pawlow³⁶, the method adopted by us has the great advantage of being identical with the standard method of gastric analysis used in clinical diagnostic work. Consequently, the results from experiments on animals obtained thereby are more easily compared with those obtained from clinical observations in man, while the

criticism referred to above lessens in importance as it may be applied to both. The small number of successful experiments presents a real difficulty in coming to definite conclusions; but, even so, it seems to us justifiable to infer from these observations the presence of inhibitory secretory fibres in the splanchnic nerves, the excision of which has a definite action in raising the secretion of HCl both in the fasting juice and in the gastric secretion after a standard test-meal.

The number of experiments has been necessarily limited, owing to the many difficulties encountered in carrying them out, and also by the time it has taken to follow the observations to the end, namely two years. The experimental work, moreover, was undertaken mainly with a view of confirming the clinical observations and deductions which have been discussed in the first division of this paper. It seems to us that this aim has been achieved, as the results, although not startling, are all more or less uniformly in agreement with one another, and we hope that they may offer an inducement to further work on this subject.

PROTOCOLS OF EXPERIMENTS ON THE ACTION OF THYROID FEEDING ON GASTRIC SECRETION.

Protocol 1.—Dog VII (weight 1 st. 5 lb., or kilo. 8.540).

A Janeway fistula and left splanchnic resection had been performed 27 days before commencement of thyroid feeding. The fractional gastric analysis used as control was taken 19 days before. Thyroid extract (Burroughs & Wellcome 'tabloid' thyroid gland) was given in the initial dose of 15 gr. thrice daily, but as it did not cause loss of weight it was increased to 45 gr. three times a day (i.e., 1 grm. of thyroid per kilo. of body weight), and at the end of a month the dog had lost 1 lb., or 5 per cent of its weight. Fractional gastric analyses were made on the 12th and 21st days of thyroid feeding.

Result.—A very definite delay of gastric secretion with lowering of the HCl curve leading to nearly a complete achlorhydria after 21 days of thyroid administration (see Fig. 248).

Protocol 2.—Dog V (weight 1 st. 11 lb., or kilo. 11.200).

A Janeway fistula and left splanchnic resection had been performed 34 days prior to the thyroid feeding. Fractional test-meal used as control was taken 25 days before. Thyroid extract administered for 12 days in the dose of 20 gr. thrice daily had no effect on the weight, and it was therefore increased to 60 gr. (i.e., 1 grm. per kilo. of body weight). After the 5th day of intensive thyroid feeding the dog had lost 8 oz. in weight, and on the 13th day it died suddenly. At the autopsy marked scarring of the kidneys was found. The fractional test-meal was taken on the 8th day of intensive thyroid feeding.

Result.—Complete achlorhydria except for the presence of HCl in the fasting juice and in the last sample (see Fig. 247).

Protocol 3.—Dog III (weight 2 st. 11 lb., or kilo. 17.472).

A Janeway fistula and excision of the left splanchnic nerve performed 3 months before thyroid feeding. The fractional test-meal used as control was taken 1 month before. An initial dose of thyroid extract (25 gr.) was given, and at the end of 4 days it was raised to 40 gr. and continued for another 4 days. At the end of this time the dog had not lost weight, so the thyroid was again raised to a maximal dose of 90 gr. thrice daily (i.e., 1 grm. per kilo. of body weight). By the end of the 2nd week of intensive thyroid medication the dog had lost 1 lb. 12 oz., and at the end of a month 2 lb. Fractional test-meals were taken on the 8th and 15th days of intensive thyroid feeding.

Result.—Apart from the absence of free HCl in the fasting juice of the tests taken after thyroid feeding there is little, if any, lowering of the gastric acidity (see Fig. 249).

Protocol 4.—*Dog VI* (weight 1 st. 8 lb., or kilo. 9.856).

A Janeway fistula and section of the left splanchnic nerve performed 34 days before thyroid feeding. Fractional test-meal used as control was taken 19 days after 9 days; the thyroid was raised to 50 gr. thrice daily (i.e., 1 grm. per kilo. of body weight), and at the end of a month the dog lost 2 lb., or 10 per cent of its weight. Fractional gastric analyses were taken on the 12th and 21st days of intensive thyroid feeding.

Result.—Absence of free HCl in the fasting juice of all the test-meals. The curve of free HCl is slightly higher in the charts of test-meals taken after the administration of thyroid (see Fig. 250).

PROTOCOLS OF EXPERIMENTS ON BILATERAL SPLANCHNIC EXCISION.

Protocol 5.—*Dog III* (weight 2 st. 11 lb.).

Dec. 1, 1926.—Excision of the left major splanchnic nerve and a Janeway fistula performed. Fractional gastric analyses were made on three occasions to determine the normal range of secretion.

Result.—The curves, although varying in detail, have some common characteristics. The fasting juice on two occasions contains bile, and free HCl is absent, while the third time the free HCl attains the value of 30 and there is a trace of bile present. The meal provokes a delayed response, the acid curve rising respectively on the three occasions $2\frac{1}{2}$, 3, $3\frac{1}{2}$ hours after the test meal, and being sustained even at the end of 5 hours. The rate of emptying is very slow, and starch is present to the end of the test, while bile is absent throughout. The curve of the total chlorides follows very closely that of the free HCl.

June 9, 1927.—General ether anaesthesia, laparotomy and excision of the right splanchnic nerve. Fractional test-meals were taken thereafter at intervals of 7 days, 3 weeks, and $3\frac{1}{2}$ months.

Result.—The charts of test-meals taken after bilateral denervation show a remarkable change from a normal chlorhydria to a definite hyperchlorhydria. On three out of four occasions free HCl was present in the fasting juice, the values ranging from 25 to 50. The meal provokes a much prompter response, the acid curve rising at once during the second hour, and attaining definitely higher values. The highest acid curves were observed 3 weeks and $3\frac{1}{2}$ months after complete denervation. The emptying time was unchanged.

POST MORTEM.—The stomach and duodenum were found to be perfectly normal, and there was no trace of ulceration or hæmorrhage, past or present. The splanchnic division was confirmed by histology of the excised portion of nerve.

Protocol 6.—*Dog VI* (weight 1 st. 8 lb.).

Jan. 19, 1927.—General ether anaesthesia, laparotomy, excision of the left splanchnic nerve, and a Janeway fistula made. A control fractional test was taken a fortnight after.

Result.—Absence of free HCl in the fasting juice, an acid curve rising after $1\frac{1}{4}$ hours, falling after $2\frac{1}{2}$ hours, and then rising again. No duodenal reflux occurred during the test, but a trace of bile was present in the fasting juice.

June 9, 1927.—Division of the right splanchnic nerve. Fractional test-meals were taken thereafter at intervals of 6 days, 13 days, and 1 month.

Result.—Except for the first meal, all show free HCl in the fasting juice, the values varying between 5 and 55. The curve rises more quickly and reaches the maximal value at an earlier stage than before denervation, and is also slightly raised. Marked duodenal regurgitation at the end of 3 hours. Emptying rate unchanged.

THE SYMPATHETIC, AND GASTRIC ACIDITY 305

POST MORTEM.—The dog died 3 months after complete denervation, of general peritonitis, caused probably by infection introduced from the fistula. Some acute flat ulcers found in the duodenum were attributed to toxic absorption secondary to the general peritonitis. Splanchnic division confirmed by histology of the excised portions of nerve.

Protocol 7.—*Dog VII* (weight 1 st. 5 lb.).

Jan. 26, 1927.—General ether anaesthesia, laparotomy, left splanchnic nerve excised, and a Janeway fistula made.

Result.—Control test-meal, taken 10 days after, showed absence of free HCl in the fasting juice and presence of bile. The acid curve rises 1½ hours after the meal, attains only low values, and then falls rapidly. There is marked duodenal regurgitation.

June 9, 1927.—Right splanchnic nerve excised.

Result.—Tests taken 12, 22, and 28 days after denervation. Absence of free HCl in the fasting juice on two out of three occasions, and presence of bile in the fasting juice. The acid curve is somewhat delayed, but maintains the same type as before denervation and there is no rise in the free HCl value.

POST MORTEM (dog killed).—Stomach and duodenum found to be perfectly normal. The division of the splanchnic nerves was confirmed both by dissection and histology of the excised portions of nerve.

Protocol 8.—*Dog VIII* (weight 2 st. 10 lb. approx.).

March 16, 1927.—General anaesthesia, laparotomy, excision of left splanchnic nerve, and a Janeway fistula made.

Result.—Control test-meal taken 10 days after: no free HCl in the fasting juice, the acid curve rises 2½ hours after the meal, and falls to fasting level 4½ hours after. There is no duodenal regurgitation.

June 9, 1927.—Right splanchnic nerve excised.

Result.—Test-meals taken 13, 22, 32, 35 days after, and 5½ months after. On two out of four occasions free HCl is present in the fasting juice, the values varying from 5 to 75. Except for one occasion the acid curve is markedly delayed, reaches a definitely higher value than before denervation, and is sustained until the end of the test.

POST MORTEM (dog killed).—Stomach and duodenum normal. The excision of the splanchnic nerves was confirmed both by dissection and by histology of the excised portions. The spleen was found to be enlarged and to contain numerous large nodular formations. We are indebted to Dr. J. S. Young for the following histological report:—

General Features.—The pulp of the organ is less cellular than normal, whereas there appears to be increase of connective-tissue stroma. Numerous multinucleated masses of protoplasm, conforming to the type of megakaryocytes, are scattered irregularly through the pulp. There is evidence of a fairly active blood destruction prior to death, in the presence of a considerable proportion of hæmosiderin. Two large nodular formations, composed principally of cells of lymphocytic type, are present in the section.

Nodular Formations.—These nodules present a delicate reticular framework, enmeshing cells of small lymphocytic type. A scanty proportion of large 'endothelioid' cells is observed, together with a relatively small number of eosinophil leucocytes: there are no giant cells, of the type which is more or less characteristic of lymphadenoma in the human subject.

DIAGNOSIS.—A lesion of this description is not uncommon in the dog, and it has been designated variously as pseudoleukæmia, Hodgkin's disease, or lymphadenia.

CONCLUSIONS.

Evidence has been brought forward to show that the sympathetic contains inhibitory secretory fibres to the stomach, and the depressive influence

of the sympathetic system on the secretion of free HCl in the stomach has been deduced from the following observations :—

1. The constant tendency towards subnormal or absent secretion of free HCl in hyperthyroidism.

2. The lowering effect of experimental thyroid feeding on gastric acidity.

3. The definite inhibitory action of adrenalin administration on gastric acidity both in man and animals.

4. The hyperchlorhydria induced by the paralytic action of nicotine on the sympathetic in man.

5. The depressive influence of emotions on gastric secretion.

6. The experimental hyperchlorhydria induced in dogs after bilateral division of the major splanchnic nerves.

Certain conclusions of clinical importance may be drawn from these observations with regard to hypochlorhydria or achylia associated with extra-gastric diseases. Although a large number of these cases may be explained by asthenia of the gastric glands in debilitating conditions (cancer, apart from carcinoma of the stomach, anæmias, fevers, etc.) or by permanent damage caused by bacterial toxins (chronic rheumatoid arthritis, chronic colitis, pernicious anæmia, etc.), others may be caused by persistent inhibitory reflexes (hyperthyroidism, gall-bladder disease). Similarly, cases of hyperchlorhydria may be explained by neuritis of the solar plexus caused by focal infections (chronic appendicitis) or by a diminished tone of central inhibitory sympathetic centres (hyperchlorhydric dyspepsia due to overwork, worry, and the ordinary stress of life).

We desire to express to Professor B. A. M'Swiney our thanks for his aid and advice.

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REFERENCES.

- ¹ LOCKWOOD, B. C., *Jour. Amer. Med. Assoc.*, 1925, lxxxv, 1032.
- ² BARKER, quoted by Lockwood, *Ibid.*
- ³ KING, quoted by Lockwood, *Ibid.*
- ⁴ WOLPE, quoted by Lockwood, *Ibid.*
- ⁵ LEIST, abstr. *Ibid.*, 1921, lxxvii, 742.
- ⁶ NEILSON, C. H., *Ibid.*, 1914, lxii, 434.
- ⁷ BOENHEIM, *Deut. med. Woch.*, 1921, xlvii, 1256.
- ⁸ MARANON, quoted by Falta, *Endocrine Diseases*, 3rd ed., 76.
- ⁹ SAJOUS, quoted by Lockwood.
- ¹⁰ MOLL, H., and SCOTT, R. A. M., *Lancet*, 1927, i, 68.
- ¹¹ BOENHEIM, quoted by Lockwood.
- ¹² ROGERS, J., RAHE, J. M., and ABLAHADIAN, E., *Amer. Jour. Physiol.*, 1919, xlviii, 79.
- ¹³ TRUESDALE, C., *Ibid.*, 1926, lxxvi, 20.
- ¹⁴ HARDT, *Ibid.*, 1916, xl, 314.
- ¹⁵ LANGLEY, *The Autonomic Nervous System*, 1921.
- ¹⁶ ELLIOTT, *Jour. of Physiol.*, 1905, xxxii, 420.
- ¹⁷ TUMPOWSKI, quoted by Lockwood.
- ¹⁸ BROWN and M'SWINEY, *Quart. Jour. Exper. Physiol.*, 1926, xvi, 1.
- ¹⁹ LIM, *Ibid.*, 1923, xiii, 79.
- ²⁰ HESS and GUNDLACH, *Pflügers Archiv*, 1920, clxxxv, 121.
- ²¹ MOLL, H., *Brit. Med. Jour.*, 1926, i, 1079.

- ²² CANNON, *The Mechanical Factors of Digestion*, 1911.
- ²³ AUER, *Amer. Jour. Physiol.*, 1907, xviii, 356.
- ²⁴ MURPHY and CANNON, quoted by Cannon.
- ²⁵ LANGDON BROWN, *The Sympathetic Nervous System in Disease*, 1923, 87.
- ²⁶ BENNET and VENABLES, *Brit. Med. Jour.*, 1920, ii, 662.
- ²⁷ BICKEL and SASAKI, *Deut. med. Woch.*, 1905, xxxi, 1829.
- ²⁸ SCHUPFER, F., *Policlinico* (Sez. Med.), 1906, xiii, 141.
- ²⁹ GAULTIER, R., *Comptes rend. Soc. de Biol.*, 1907, lxii, 865.
- ³⁰ DUCCESCHI, *Arch. Fisiol.*, 1905, ii, 525.
- ³¹ MCCREA, M'SWINEY, and STOPFORD, *Quart. Jour. Exper. Physiol.*, 1926, xvi, 195.
- ³² CARLSON, *Physiol. Reviews*, 1923, iii, 1.
- ³³ BELL, J. R., and MACADAM, W., *Amer. Jour. Med. Sci.*, 1924, clxvii, 4, 520.
- ³⁴ LIM, MATHESON, and SCHLAPP, *Edin. Med. Jour.*, 1923, July, 265.
- ³⁵ BAIRD, CAMPBELL, and HERN, *Proc. Physiol. Soc., Jour. of Physiol.*, 1923, lviii, 20.
- ³⁶ PAWLOW, *The Work of the Digestive Glands*, 2nd ed., 1910, London.
- ³⁷ LIM, R. K. S., *China Med. Jour.*, 1925, June.
- ³⁸ BOLDYREFF, W., *Quart. Jour. Exper. Physiol.*, 1914, vii, 7.
- ³⁹ BOLTON and GOODHART, *Lancet*, 1922, i, 420.
- ⁴⁰ BAIRD, CAMPBELL, and HERN, *Guy's Hosp. Rep.*, 1924, lxxiv, 37.

SPECIAL ARTICLES
ON SURGICAL TECHNIQUE.

ON THE REMOVAL OF CEREBRAL TUMOURS.

By SIR PERCY SARGENT, C.M.G., D.S.O.,
SENIOR SURGEON TO ST. THOMAS'S HOSPITAL, LONDON.

THE true neoplasms which involve the cerebral hemispheres, and which can be wholly or partly removed, are either extra-neural or intra-neural. The former are, with rare exceptions, of meningeal origin, and are usually encapsulated tumours belonging to the group termed endotheliomata. Although they may invade the overlying bone they never infiltrate the brain, but lie



FIG. 269.—Endothelioma, showing the encapsulation, and the clearly defined bed in which the tumour lies.

loosely embedded in cup-like cavities from which they can be cleanly and completely enucleated (*Figs. 269, 270*).

The intra-neural tumours belong to the group generically termed gliomata, of which there are many varieties, differing in structure and clinical behaviour. The degree of delimitation of the gliomata varies greatly, but they are never

truly encapsulated (*Fig. 271*). It follows, therefore, that a glioma can rarely be removed with the same completeness as an endothelioma. This, however, should not deter the surgeon from making as determined an attack upon a favourable glioma as the circumstances warrant, having regard always to the



FIG. 270.—An endothelioma removed by operation, together with the overlying and adherent dura.



FIG. 271.—Glioma, showing moderately definite delimitation without encapsulation.

function of that part of the brain which is involved. For example, a radical extirpation might be legitimate in the occipital region which would be unpardonable in the areas concerned with movement or speech.

I am not here concerned with the technique of cerebral exploration in

obscure cases, nor of simple decompression, but with the procedures which may be adopted when a reasonably certain diagnosis has been arrived at regarding the presence and situation of a tumour in one of the cerebral hemispheres. It is more often possible to be sure on these points than as to the nature and size of the tumour, so that, with the exception of the few cases in which an endothelioma can be demonstrated by the X-ray appearances, every operation must of necessity be primarily of an exploratory character. The technique of exploration, therefore, must be carried out with a view to possible removal.

In the first stage—the exposure of the tumour—the operative technique is naturally the same as for all intracranial procedures.

GENERAL PRINCIPLES.

Anæsthesia.—My own preference is for general anæsthesia with ether, administered by the intratracheal route. It cannot be denied that local anæsthesia offers some advantages, but these are, in my opinion, outweighed by one's distaste for operating upon a conscious patient, by the extra time expended, and by the possibility of tissue necrosis. If a patient is so deeply narcotized with morphia as to suffer no physical inconvenience or mental distress, it is a misuse of language to call the anæsthesia 'local'. In my experience intratracheal ether fulfils almost all the requirements of an ideal anæsthetic in cerebral surgery. The patient can be placed in any position which the operator may find most convenient; the dosage of ether can be regulated as required at the several stages of the operation; oxygen or carbon dioxide can be given if and when necessary; and the anæsthetist is out of the way. Chloroform is dangerous, particularly in the presence of increased intracranial pressure, and should rarely if ever be employed.

Control of Hæmorrhage.—Hæmostasis is important, but its importance can be exaggerated, and if such exaggeration leads to waste of time, the additional exposure and prolonged anæsthesia must go far to counteract the advantage of saving a little blood.

In cerebral operations the arrest of arterial bleeding presents no difficulties save in some rare cases of meningeal tumour of excessive vascularity. It is the venous bleeding which is most embarrassing, and may even be dangerous. The ordinary venous bleeding is readily kept in check by:—

1. *Posture.*—The head should always be raised well above the level of the trunk in all intracranial operations, and at the same time care should be taken that the jugular veins are not compressed.

2. *Oxygenation of the Blood.*—This diminishes venous pressure and checks capillary oozing.

3. *Continuous Irrigation with Hot Saline.*—The fluid may be at 120° F. during the earlier stages, but the temperature should be somewhat lowered after the dura is opened. This irrigation diminishes loss of blood, saves time, and keeps the field clear. It is the best way of 'swabbing' the delicate tissues.

SPECIAL MEANS OF CONTROLLING AND ARRESTING HÆMORRHAGE.—

1. *Special Forceps for the Scalp.*—Ordinary Spencer-Wells forceps are inconvenient and inefficient. With them it is difficult to pick up quickly all

the individual bleeding points, especially on the concave side of the flap. The forceps which I use clamp lightly and harmlessly the whole thickness of the scalp, irrespective of the position of individual vessels; they can be applied very quickly, and lie out of the way, leaving the operative field clear. For the convex side of the flap I use curved toothed forceps. By using these instruments the whole scalp incision can be completed in two or three minutes, and with trifling loss of blood.

2. *Horsley's Wax for the Bone*.—Small pellets of this wax, softened to a convenient consistence, are pressed into the bleeding bony openings. It should not be used too freely in cases where an osteoplastic flap is to be replaced, as it interferes with bony union.

3. *Horsley's Muscle Graft*.—If a fragment of muscle is gently pressed against a bleeding point for a few seconds, it adheres quickly, and arrests the hæmorrhage most efficiently. Even the bleeding from a tear in a large sinus can be readily controlled by this means.

4. *Fine Ligatures* carried in a slender round-bodied needle can be used for some bleeding points in and upon the brain, or *Cushing's silver clips* may be employed for the same purpose.

5. *Temporary Tamponade with Small Pieces of Gauze or Wool*.—For this purpose the rolls of wool used by dental surgeons are convenient. One of these, attached to a long thread to guard against being left behind, is pressed gently against the bleeding point and left there whilst other steps of the operation are being proceeded with. When, after a few minutes, the plug is removed, the bleeding will nearly always have ceased.

Opening of the Cranium.—Whenever any reasonable prospect exists of removing the tumour, the osteoplastic flap is employed. The actual method of cutting the bone is of little importance, and individual surgeons will use the instruments which suit them best. The essential points are that the flap should be so fashioned, so placed, and of such size as to afford the freest possible access, and to ensure the largest possible exposure of the part of the brain to be explored. Descriptions of the technique employed by different operators abound in the literature.

Intracranial Pressure.—Although symptoms of increased intracranial pressure may have been, even with large tumours, absent or inconspicuous, it is very rare indeed to find, on exposure of the dura, that the intracranial pressure is not raised. The surgeon must therefore be prepared to find that the brain bulges when the dura is opened. Unless this bulging is so great that the cortical vessels are obstructed at the margin of the dural opening—unless, in fact, the bulging brain is strangulated—the brain will suffer no harm, whilst the relief of pressure afforded results in a diminution of venous congestion generally. If, however, the pressure is so high as to endanger the brain or to hamper manipulation, steps must be taken to reduce it. The danger of lumbar puncture in the presence of increased intracranial pressure is well known, particularly in the case of tumours in the posterior fossa. When, however, the skull is opened, and the surgeon wishes to reduce the pressure, lumbar puncture may be safely employed. An attempt also may be made to reach and remove fluid from the ventricles, but the lateral ventricle is usually obliterated on the side of the tumour. In searching for

it with an exploring needle, however, a gliomatous cyst may happily be encountered.

Examination of the Exposed Brain.—When the point of attachment of an endothelioma to the dura lies beneath the bone-flap, it will be recognized at once by its umbilicated appearance, and the tumour can be felt. In such a case the dura should be incised round, and at a little distance from, the point of attachment, as the adherent dura must be removed with the tumour (*see Fig. 270*). When no indication of any tumour can be seen or felt, the dura must be freely opened. This is best done by a series of radial incisions from centre to periphery of the exposed dura. By this method a number of triangular flaps of dura are fashioned, each with its base at the margin of the bony opening, so that there is no risk of the brain being damaged against the cut edge of the bone.

The exposed brain presents appearances which are quite characteristic of the various conditions which may be found.

If a *meningeal tumour* is present but its surface is so situated as not to lie in the exposed area, the convolutions will give the appearance of being crowded together, whilst the sulci are displaced. Unless the pressure is inconveniently high, which is unusual in cases of meningeal tumour, it is quite easy to inspect almost the whole of the external surface of the hemisphere in the following manner. The dural flaps are successively pulled over the edge of the bone by an assistant, whilst the surgeon inserts a glass spatula between brain and dura. By depressing the spatula and with illumination from a head-light, the surface of the brain can be inspected. Should a tumour be thus exposed, the bony opening must be enlarged in order to expose it fully. A glioma infiltrating the cortex may be revealed by the same means.

The appearance of the brain over a *subcortical glioma* is quite different, and quite characteristic. The brain appears dry, the convolutions are broad, pale, and of a yellowish hue, the sulci are obliterated, the cortical veins small, and the main fissures may be displaced. For example, with a temporal glioma the Sylvian fissure is often arched upwards for a considerable distance above its natural position, whilst the whole lobe gives the appearance of being more bulky than usual.

Palpation of the brain gives information of the greatest value. It need hardly be said that this examination must be conducted with the utmost gentleness. Over a subcortical tumour the brain feels softened, and mostly so where the glioma comes nearest to the surface. The sensation conveyed to the finger cannot be described in words, but it is, in my experience, quite characteristic. A slender narrow blunt instrument passed into the brain at the softest spot will encounter and be brought up against a glioma, as this is almost always of a somewhat firmer texture than brain tissue. Should a gliomatous cyst be present and the instrument pass into it, the characteristic yellowish fluid will escape through the puncture.

REMOVAL OF TUMOURS.

Endothelioma.—*Fig. 272* indicates, in a semi-diagrammatic manner, the relationship of the endothelioma to the surrounding brain and membranes. Except at the umbilicated point of attachment it lies beneath and free from

the arachnoid. Any attempt to enucleate it with the arachnoid intact must result in serious damage to the surrounding brain and pial vessels, so that the first essential step is to incise the arachnoid round the edges of the brain-cup in which the tumour lies. As soon as this has been done, and especially if the pressure is at all raised, the tumour begins slowly to be extruded, and with a little gentle pulling and coaxing it can usually be removed from its bed cleanly and easily with insignificant bleeding.

This description applies to the easiest possible case, like that from which the illustration was taken. Unfortunately the point of attachment is more

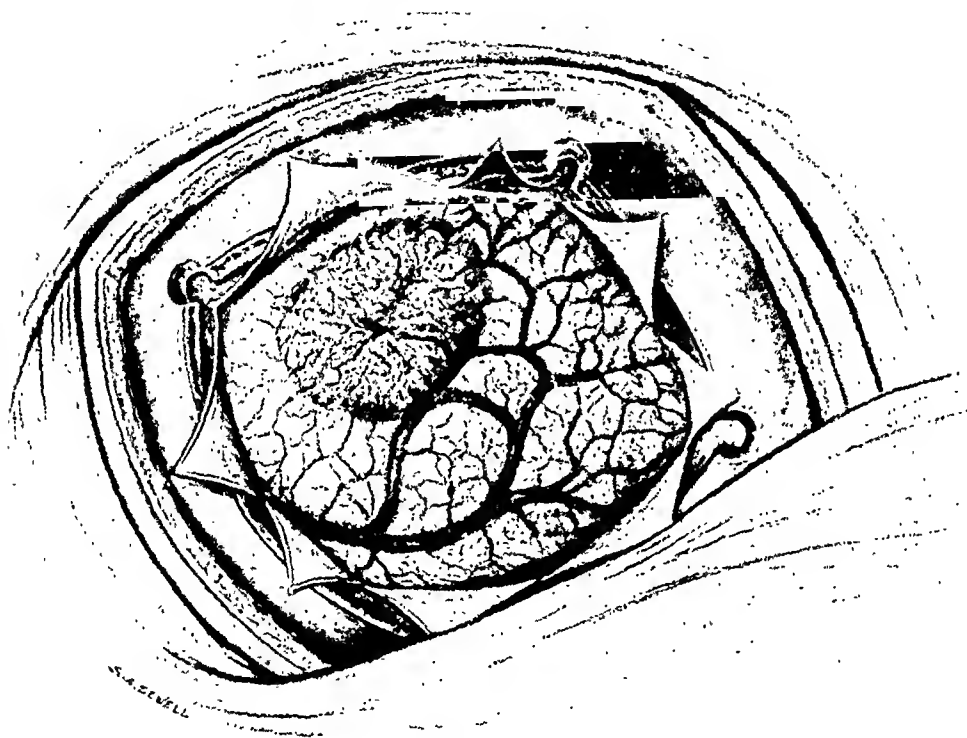


FIG. 272.—Exposure of an endothelioma. The figure shows the relation of the surface of the tumour to the arachnoid and to the surrounding convolutions. Note the clearly defined edge of the cup; also the umbilicated central point of attachment, from which the dura has been removed. (From a sketch made at operation—semi-diagrammatic.)

often near to and closely connected with the sagittal sinus, in which case the bleeding may be severe, whilst some of these tumours are so vascular that the hæmorrhage may be quite alarming. I have never, however, myself encountered a case in which it could not be satisfactorily dealt with by the means indicated in a preceding paragraph. There are few bleeding points which cannot be controlled by a Horsley's muscle graft, whilst oxygenation of the blood, hot irrigation, and temporary tamponade, patiently persisted in, will enable the most alarming bleeding to be successfully arrested. Recently Harvey Cushing¹ has been using a method of electro-coagulation for the arrest of hæmorrhage, which promises to be of value in some cases.

Glioma.—In some cases the growth is firm in texture, and more or less circumscribed. Such tumours should be removed if their removal would not necessarily leave the patient hopelessly crippled, as, for example, in the regions concerned with speech and with movement. In such cases it is best to make no attempt at removal, but to convert the operation into one of simple decompression, and to treat the tumour subsequently by radiotherapy.

In a favourable case a free incision should be made through the cortex, after ligation of any prominent vessels that cannot be avoided by the incision. The tumour being thus exposed, a blunt curved instrument can be gently passed between it and the surrounding white matter of the brain. The instrument which I have found best is that which was employed both by Horsley and by Ballance, namely, the handle of an ordinary teaspoon. A finger, if small and gentle, may be used, and more information can be gained by it than by any instrument, but naturally rather more damage must be done to the surrounding brain. The bleeding caused during the removal of a subcortical glioma is usually insignificant, and can easily be controlled by hot irrigation and temporary tamponade of the cavity.

After removal of a tumour from a cerebral hemisphere, the cavity which it occupied very quickly diminishes in size, especially when the pre-operative intracranial pressure has been high. What remains of the cavity at the completion of the operation—at first filled with saline, cerebrospinal fluid, and blood—gradually becomes obliterated.

Closure of the Wound.—No special technique is necessary for this stage of the operation. The dural flaps are merely replaced over the surface of the brain; they should never be sutured. The bone-flap is fitted carefully into position, and the scalp accurately sutured.

REFERENCE.

- ¹ CUSHING, HARVEY, *Macewen Memorial Lecture*, 1927.

*SHORT NOTES OF
RARE OR OBSCURE CASES*

HYPERTROPHY OF THE GUMS.

By R. J. McNEILL LOVE,

SURGEON TO ST. ANDREW'S HOSPITAL, LONDON, E.

J. C., age 18, was admitted to St. Andrew's Hospital, Poplar, with the following history: Three years ago, the gums of both jaws began to increase in size. This caused little disability at first, but after about a year it was noticed



Fig. 273.—Skiagram showing irregular and displaced teeth, and normal sella turcica.

that speech was somewhat thick and indistinct. The enlargement of the gums was progressive, and one year ago it was difficult to cover them with the lips, so that in moments of repose the gums protruded from the mouth between the half-closed lips. Further growth occurred, and the deformity

became so grotesque that any appearance in public was shunned. Finally the patient kept indoors continually, except at night.

ON EXAMINATION.—The superior alveolar border appeared as a U-shaped pad, the lower being V-shaped. These pads were firm and nearly 1 inch in breadth, being particularly thick and broad in the region of the tuberosities of the superior maxilla. Two or three infected stumps were visible on the surface. The lower jaw appears to be somewhat prognathous, and the hands and feet are undoubtedly enlarged.

X-ray examination showed marked displacement of the teeth, which were completely submerged on the surface of the pads representing the gums. There was no enlargement of the sella turcica (*Fig. 273*).



FIG. 274.—Photograph of patient after removal of the alveolar border of the upper jaw, showing protrusion of the lower hypertrophic gums. The tongue can be distinguished behind this mass. Enlargement of the hands is also shown.

OPERATION.—The patient was admitted to hospital in September, 1927, and the hypertrophic gums together with the teeth were removed in stages with the chisel, excessive hæmorrhage being controlled by diathermy (*Fig. 274*). Healing occurred without event. Section of the gum shows fibrous overgrowth, with no evidence of excessive lymphatic tissue.

The enlargement of the lower jaw and extremities suggests acromegaly, but no other signs or symptoms of this condition are present.

I am indebted to Dr. Eric Hardy, of St. Andrew's Hospital, for assistance with radiography and microscopy.

GIANT RENAL CALCULUS WITH EPITHELIOMA IN A HORSESHOE KIDNEY.

By R. J. WILLAN, M.V.O.,

HONORARY SURGEON AND LECTURER IN CLINICAL SURGERY TO THE ROYAL VICTORIA INFIRMARY,
NEWCASTLE-UPON-TYNE.

IN April, 1927, Professor Thomas Beattie asked me to see, in consultation, J. H., age 56, a male patient under his care at the Royal Victoria Infirmary,



FIG. 275.—Showing a large dense shadow in the area of the swelling. (*Skiagram reversed.*)

Newcastle-on-Tyne. At the age of 20 there was a history of a seven months' illness, when he was in bed for six weeks; he had no pain, and he was told he had suffered from 'nephritis'. In May, 1926, he had painless hæmaturia for three days; from that date up to the time of admission there had been increased frequency of micturition, while the urinary stream was poor.

ON ADMISSION.—He complained that he had severe pain in the right ilio-costal space radiating to the thigh, that he was getting thinner, that his urine was discoloured, and that he had increased frequency of micturition.

There was a visible abdominal swelling to the right of the umbilicus. On palpation, a swelling the size of a hen's egg could be felt behind the right rectus muscle at the level of the umbilicus. The swelling was hard, smooth, had well-defined margins, and did not move on respiration. X rays (*Fig. 275*) showed a large dense shadow in the area of the visible and palpable swelling. His urine contained albumin, blood, pus, also triple phosphates. *Bacillus coli communis* was obtained on culture. No other abnormal physical signs were elicited.



FIG. 276.—Double pyelogram; absence of any communication between the renal pelves. (*Skiagram reversed.*)

Owing to the unusually low level of the X-ray shadow I thought there might be a horseshoe kidney, and I considered it probable that pyelography would confirm or refute the suggestion. Double pyelography (*Fig. 276*) failed to show any communication between the renal pelvis of each kidney, and the suggestion of horseshoe kidney was regarded as erroneous. The left kidney renal function being efficient, an operation was advised.

OPERATION.—A horseshoe kidney was found. An attempt to open the right renal pelvis posteriorly failed owing to dense adhesions, but an incision through the anterior aspect of the pelvis enabled a large calculus to be removed. The pelvis contained a considerable amount of purulent debris and blood-

clot; its cut margins were approximated with interrupted catgut sutures. The removed calculus weighed 117.2 grm. (or 4 oz. 80 gr.). The patient stood the operation well and there was not much constitutional disturbance, but he died the following day from an unexpected sudden heart attack.

POST-MORTEM.—A post-mortem examination was made, and through the

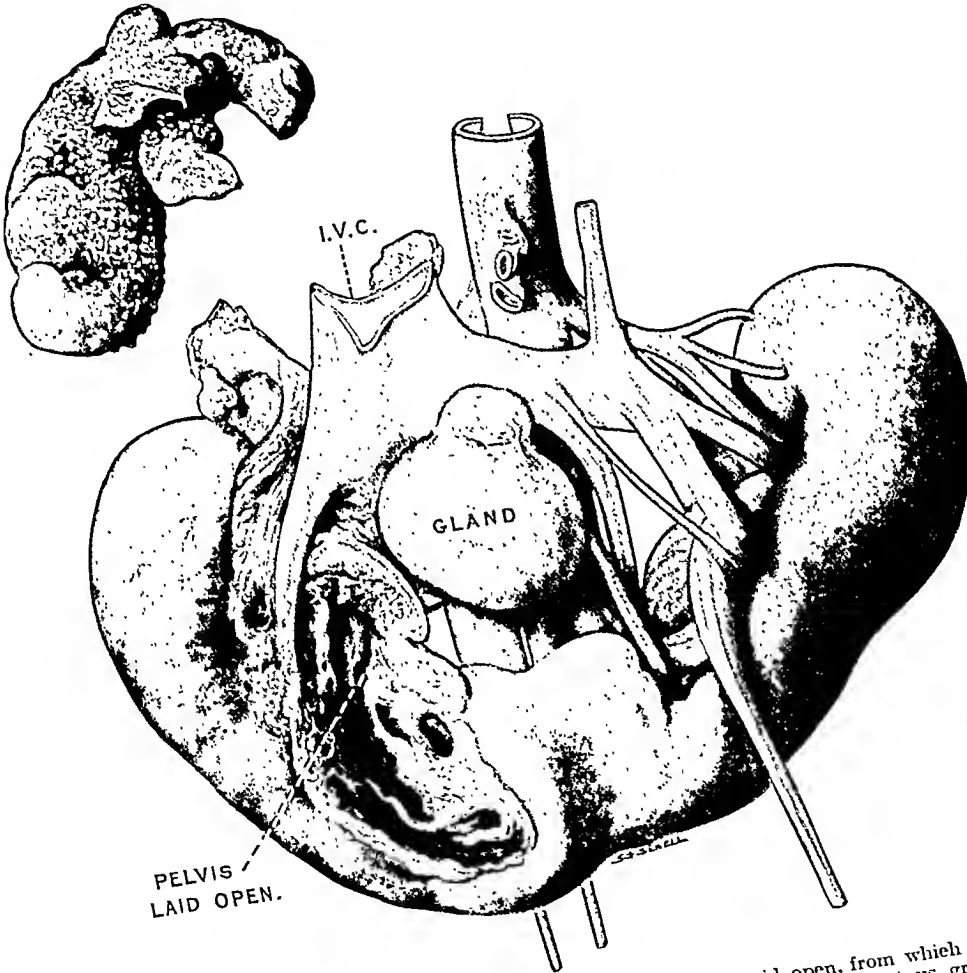


FIG. 277.—Front view, showing the right renal pelvis laid open, from which the large calculus (see upper part of picture) was removed. Necrotic epitheliomatous growth can be seen in the upper part of the right pelvis.

courtesy of Professor Stuart MacDonald I was able to obtain the specimen. It is a horseshoe kidney with fusion of the lower poles. Its relation has been preserved to the aorta, inferior vena cava, the renal vessels, and the left ureter. The front view of the specimen (Fig. 277) shows a normal-looking left

pelvis and ureter. The anterior part of the right pelvis with the ureter have been removed; both ureters lay in front of the bridge of renal tissue. In the right pelvis there is a necrotic mass of growth which extends into the upper part of the right kidney substance, also up behind the inferior vena cava. A large malignant aortic gland is seen in anterior relation to the large vessels. The back view of the specimen (*Fig. 278*) shows the large vessels laid open.

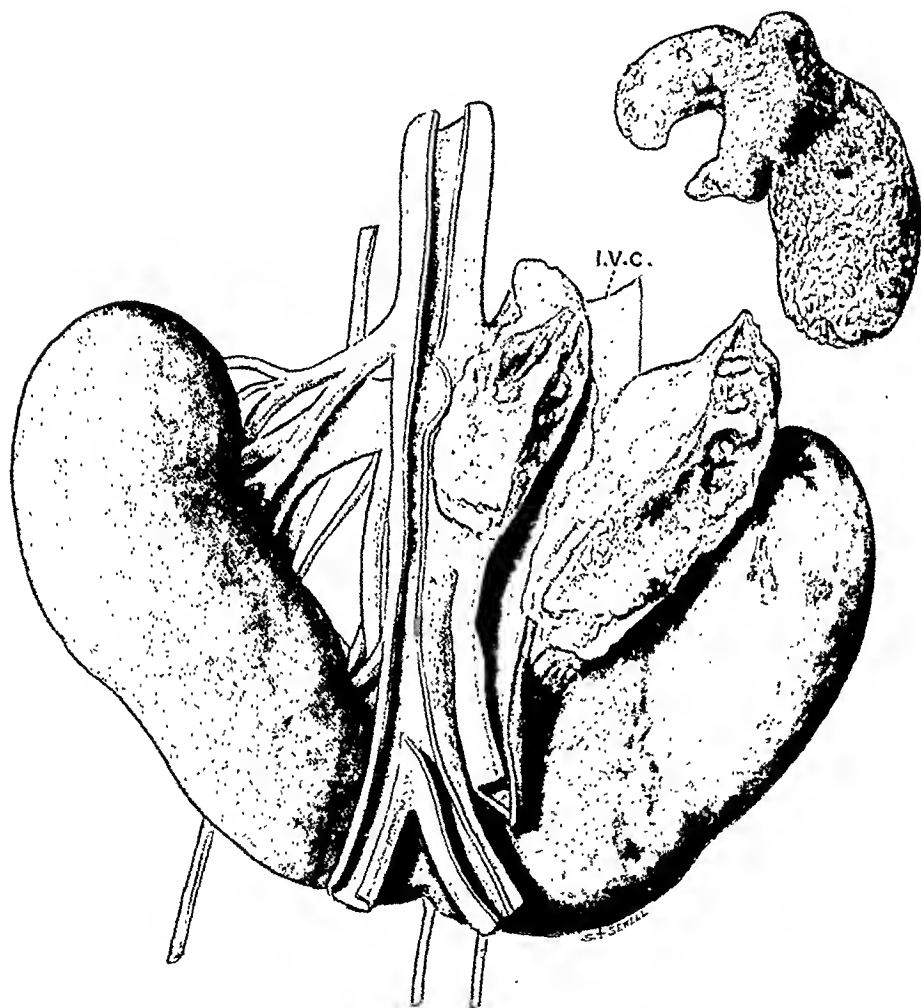


FIG. 278.—Back view, showing the malignant growth in relation to the great vessels and posterior surface of the right kidney; also the calculus.

The malignant growth involves the aortic lymph glands and the right suprarenal capsule. The calculus removed at operation is shown in the upper part of *Figs. 277, 278*.

A more minute inspection of the specimen showed that in the left half of the horseshoe kidney there was an early stage of acute pyelitis.

A microsection (*Fig. 279*) shows a definite carcinomatous condition infiltrating the kidney substance from the pelvis. The cells are epithelial and of transitional or squamous type.



FIG. 279.—Microscopic section showing the carcinoma invading the kidney substance.

I am indebted to Professor Stuart MacDonald for the pathological slide, and to Dr. H. E. Gamlen for the radiograms.

A CASE OF SQUAMOUS CARCINOMA OF THE SCALP, ASSOCIATED WITH TWO RODENT ULCERS ON THE SHOULDER ORIGINATING IN A PATCH OF PSORIASIS.

By E. R. FLINT AND J. GORDON,

THE GENERAL INFIRMARY, LEEDS, AND THE DEPARTMENT OF PATHOLOGY AND BACTERIOLOGY
IN THE UNIVERSITY OF LEEDS.

CLINICAL HISTORY.—The patient, age 59, a foreman painter, two years ago received an injury to the top of his head, causing a small wound which began to discharge after a few days. The wound continued to discharge until about three months before operation, when a small lump made its appearance, and gradually increased in size. At the time of operation it was about the size of a small walnut, not very hard, and the surface was ulcerated and bled freely on being touched. The surrounding tissues were not affected apparently, and there was no enlargement of lymph glands. The growth of the scalp was excised and X-ray treatment given.

On the right shoulder within an inch of one another in the midst of a patch of psoriasis, there were present two rodent ulcers of two years' duration. The psoriasis was of a chronic type, having been present for twenty years. The rodent ulcers were about the size of a shilling and not hard. One was excised, and the other treated by X rays, to which it speedily responded.

HISTOLOGICAL EXAMINATION.—*The tumour of the scalp* was a squamous carcinoma of great cellularity and comparatively slight keratinization. It measured 15 mm. in diameter and was about 7 mm. deep. It showed marked invasion of the corium by long slender columns of cells, but nearer the surface the carcinomatous masses were large and were undergoing central necrosis. A few keratinous cell-nests were present. The stroma was everywhere abundant, and there was lymphocytic reaction at the margins of the tumour, but none on its deep aspect. The surface was ulcerated, with some small shreds of purulent crust still adhering. On one side of the tumour a few detached islets of neoplastic tissue were seen, at least 4 mm. from the general spreading edge. The tumour was obviously a frankly malignant squamous carcinoma and was tending to infiltrate widely.

The tumour from the shoulder was a typical basal-celled carcinoma of the solid massive type. At the periphery of the epithelial masses there was a single layer of tall columnar epithelium. The interior was wholly made up of small oat-shaped cells between which intercellular prickles could sometimes be made out. It was ulcerated on the surface, and the stroma was the seat of considerable inflammatory reaction, being overrun by lymphocytes and plasma cells. There was a certain amount of central necrosis in some of the larger tumour masses, but nowhere any evidence of keratinization. The tumour was quite superficial and did not tend to infiltrate.

The interest of this case is twofold. Firstly, the occurrence of two types of malignant disease in the same patient. That this is not as infrequent as may be supposed is shown by Owen¹, who in reviewing 3000 cases of malignancy found twenty cases of squamous carcinoma occurring in conjunction with basal-celled carcinoma. The second point of interest is the occurrence of rodent ulcers on a patch of psoriasis. Whitfield² recorded a case of psoriasis associated with rodent ulcer, and Gray³ described a rodent ulcer occurring in a patch of psoriasis in the gluteal cleft. Sequeira⁴ states that he has had several cases under his care of multiple superficial rodent ulcers arising in patches of psoriasis.

REFERENCES.

¹ OWEN, L. J., *Jour. Amer. Med. Assoc.*, 1921, lxxvi, 1329.

² WHITFIELD, *Brit. Jour. Dermatol.*, 1906, xviii, 40.

³ GRAY, A. M. H., *Ibid.*, 1912, xxiv, 325.

⁴ SEQUEIRA, *Diseases of the Skin*, 4th ed., 1927, 558. London: J. & A. Churchill.

**PEDUNCULATED EXTRA-GASTRIC LEIOMYOMA OF THE
STOMACH WITH HÆMORRHAGIC DEGENERATION :
AN UNUSUAL CAUSE OF ACUTE INTRA-ABDOMINAL HÆMORRHAGE.**

By T. McW. MILLAR.

FROM THE DEPARTMENT OF CLINICAL SURGERY, UNIVERSITY OF EDINBURGH.

THE following case seems worth recording because of the difficulty in diagnosis and the rarity of the pathological condition found.

W. C., age 37, a printer by trade, was admitted to the Edinburgh Royal Infirmary on August 21, 1927, complaining of pain in the upper abdomen.

HISTORY.—On the morning of admission he had risen at his usual hour, feeling quite well, and had eaten a hearty breakfast. He went to work at 8 a.m., and noticed nothing amiss till about 10 a.m., when he became aware of a vague discomfort in the epigastrium. This gradually got worse. He felt as if his stomach were blown up with gas, and he became somewhat nauseated but did not vomit. In spite of these symptoms he continued hard at work till about midday, when, thinking he might get relief if his bowels moved—though he had no desire to defæcate—he went to the lavatory. While straining at stool he was suddenly seized with a severe pain in the epigastrium (? right side). He felt sick, shivered, and perspired freely, and, feeling he was about to faint, he hurriedly left the lavatory to get assistance. He fainted immediately he got outside but was unconscious only for a few minutes. He was assisted to his feet but still felt giddy, and after resting for a short time he went home, about 12.30 p.m. He lay down at home and had a cup of tea but no dinner, and, feeling a little better, decided to return to his work about 1.45 p.m. The pain in the epigastrium was still present and he noticed that the slight jarring caused by walking downstairs hurt him particularly. About 3 p.m. the pain suddenly became more severe and assumed an intermittent, stinging character. Its severity was such that it sometimes doubled him up, and he was compelled to give up work and go home about 4 p.m. He sent for his doctor, who arrived about 6 p.m. The latter, after consultation with his partner, decided to send him into hospital. Meantime the pain continued, and before leaving for his sixteen miles' journey to hospital he was given $\frac{1}{2}$ gr. heroin.

PREVIOUS HEALTH.—Patient had had no previous illness of importance. He had malaria and a slight attack of dysentery while on active service. All his teeth were removed three years ago because of root abscesses. He had a short-lived attack of severe pain in the stomach six months ago, since when he had remained quite well until two weeks before admission, when he began to feel out of sorts, and both he and his wife thought he was looking paler than usual.

ON EXAMINATION.—Temperature 98.4°, pulse-rate 88, respiration 20. The patient appeared to be comparatively comfortable, and was perspiring slightly. He stated that he had felt much easier since he had had the hypodermic injection. The abdominal wall on inspection was not moving freely,

but the abdomen showed no abnormal contour and no distention. On palpation, there was definite rigidity, but not boarding, of the upper half of both recti, and there was diffuse tenderness over the same area. Slight tenderness in the lower right quadrant. No definite hyperæsthesia. On percussion, the note was tympanic all over the abdomen except for an area of indefinite dullness over the outer half of the left rectus muscle above the umbilicus. No dullness was made out in the flanks. No change in liver dullness and no discoverable enlargement of splenic dullness. A diagnosis was made of probably leaking gastric or duodenal ulcer, with localized peritonitis (it was recognized that the indefinite dullness on the left side could not easily be explained on this diagnosis).

OPERATION.—General anæsthesia. The abdomen was opened by a right paramedian incision above the umbilicus, and the peritoneum was found to be full of bright-red, fluid blood. A rapid examination of the stomach and

duodenum revealed no perforation, and the character of the fluid at once ruled out perforation of any hollow viscus. Bleeding appeared to be active. Palpation of the liver and spleen showed these organs to be normal, but on feeling for the spleen, a large, soft, apparently cystic tumour was encountered on the left side. Attempts to bring it up to the wound showed that it was fixed posteriorly, and it was at first thought it might be a tumour of the left kidney. On passing the hand over the lateral side of the tumour, however, the left kidney was found to be normal. It was then realized that the tumour was in the lesser sac, and to obtain better access the left rectus muscle was cut across at a point opposite the middle of the first incision. Bleeding still seemed to be active. The lesser sac was then opened through the gastrocolic omentum, and the tumour brought to view. It was about the size of a large Jaffa orange, and was found to be fixed to the posterior wall of the lesser sac. While carrying out an examination of the tumour its wall ruptured and a large quantity of dark semi-solid material which appeared to be old

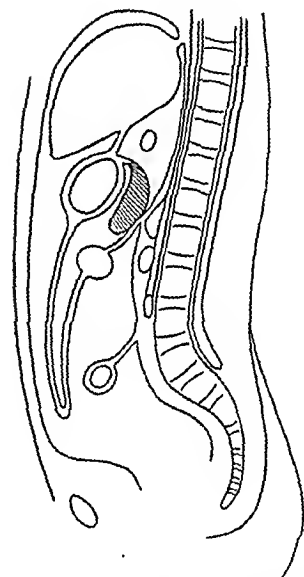


FIG. 280.—Sagittal section of the abdominal cavity to show the position and relations of the tumour (shaded).

blood-clot was evacuated. The cyst wall that remained was then examined from within the cyst, and several large dilated venous channels were seen in the wall. On examining it again from the outside, in its now collapsed condition, it was found, on tracing it upwards, to taper off to a thin, rounded, solid pedicle, about $\frac{1}{4}$ in. thick, by which it was attached to the posterior wall of the stomach at a point $\frac{3}{4}$ in. below the lesser curvature and almost opposite the mid-point of the latter (Fig. 280). This gastric attachment was clearly seen, and it was then evident that we were dealing with a pedunculated external tumour of the stomach, of unknown type; but it could not even then be determined where the blood was coming from. Acting on the assumption that the tumour—being the only pathological condition

found—was the source of the bleeding, it was decided to remove as much of it as possible. Total removal of the cyst wall could not be carried out because of its strong adhesion over a considerable area of the posterior wall of the lesser sac, and because of the great danger of injuring important structures in the region, notably the left branch of the middle colic artery. Accordingly

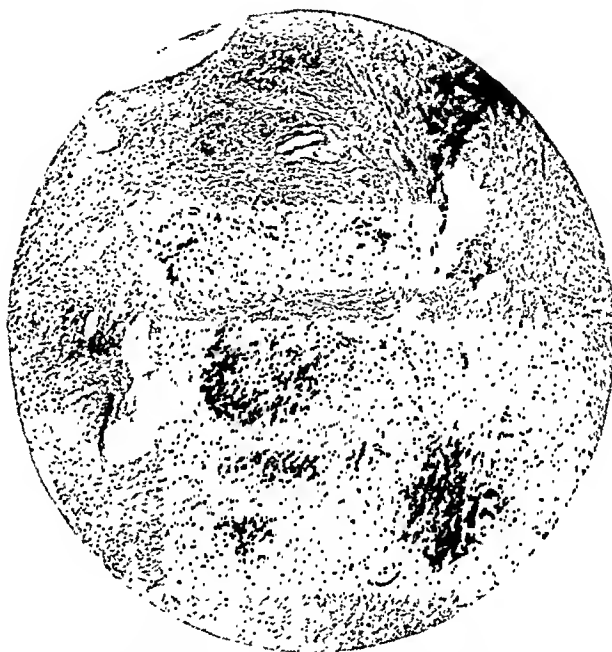


FIG. 281.—Section of pedicle of cyst showing masses of tumour tissue amongst the muscular fibres from the stomach wall.

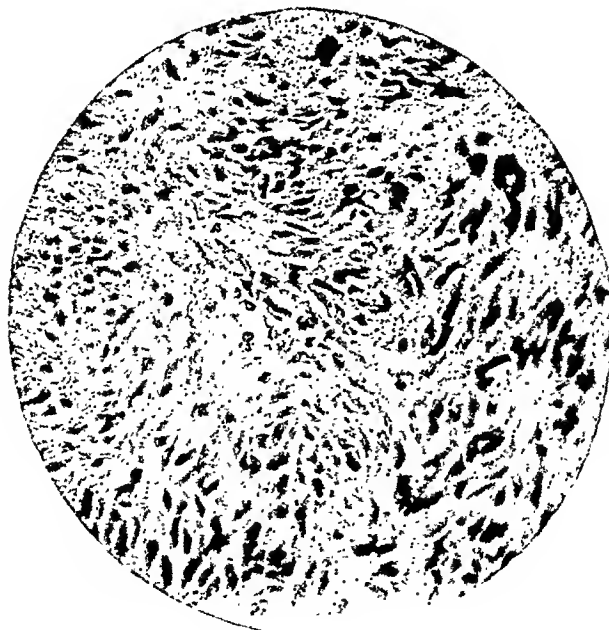


FIG. 282.—High-power view showing type of cell of which the tumour was composed.
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the pedicle was divided between clamps, close to the stomach wall, the stump ligated and overstitched, and as much of the wall as possible removed, the cut edges of the portion left behind being ligated. On now swabbing out all the blood in the abdominal cavity, it was seen that the hæmorrhage had been completely controlled. The abdomen was therefore closed as rapidly as possible and the patient returned to bed. He had lost a considerable quantity of blood and his condition was none too good; it was therefore decided to give him an immediate blood transfusion. His wife, who had come to hospital with him, was fortunately found to be a Group IV donor, and a pint of blood was obtained from her and given to the patient. He made an excellent recovery. His temperature was a little unstable for six days, but then settled completely. His condition caused no further anxiety, and he left hospital on the 21st day after admission. When seen recently he was perfectly well and had returned to work.

PATHOLOGY.—Part of the pedicle and wall of the cyst were examined microscopically. In the pedicle was found normal muscle from the stomach wall together with areas of leiomyomatous tissue interspersed between the muscle fibres. In the section of the wall considerable hæmorrhages in various stages of organization were seen, and here and there little remnants of tumour tissue, again leiomyomatous in nature (*Figs. 281, 282*).

REMARKS.

Simple tumours of the stomach are comparatively rare, the commonest being the myoma or fibromyoma. They may be external or internal. If external, they are usually symptomless until they attain a considerable size, when they may give rise to symptoms due to their weight or bulk dragging or distorting the stomach. Occasionally they grow to a great size (e.g., in Von Erlach's case the tumour weighed 5400 grm.). They are liable to various forms of degeneration—myxomatous, hyaline, hæmorrhagic, or malignant. When repeated hæmorrhages occur into the tumour it tends to become cystic, as in the present case. The writer has not seen a case reported in which a large intra-abdominal hæmorrhage was brought about. In the case described, the exact source of the hæmorrhage was not found, and although large dilated veins were seen in the cyst wall, it was noticed that the thin pedicle showed no obvious vessels. It would therefore seem probable that the tumour had developed an adventitious blood-supply from some vessel or vessels in the posterior wall of the lesser sac, to which it had become adherent.

A correct pre-operative diagnosis in such a case is hardly to be looked for, although a diagnosis of intra-abdominal hæmorrhage might have been expected. It is, however, a notable fact, well illustrated by this case, that in intra-abdominal hæmorrhage the pulse-rate may be very little raised and the patient show little sign of hæmorrhage until the abdomen is absolutely full of blood. The pulse-rate may then rise suddenly and rapidly and the patient develop the typical clinical signs of severe hæmorrhage.

In conclusion, I wish to express my thanks to Professor Fraser for permission to publish the case and for helpful advice in preparing it for publication.

CYSTIC DILATATION OF THE COMMON BILE-DUCT: RECORD OF AN EXAMPLE.

By JULIAN TAYLOR,

ASSOCIATE SURGEON TO UNIVERSITY COLLEGE HOSPITAL, LONDON.

At the present time (April, 1928) it appears from perusal of the most recent publication referring to this malady¹ that, whereas it has once been recorded as recognized before operation in a fatal case,² there is no published account of a correct diagnosis followed by successful operation. The following is the record of such a case. The writer is indebted to the bibliographic tables of the American authors for a summary of the literature of the disease, and to a substantially similar collection made recently by Professor Choyce, Director of the Surgical Unit of University College Hospital, that he has been privileged to read in manuscript.

CLINICAL HISTORY.—N. P., female, age 23, was seen by the writer in consultation with Dr. Charles Bolton on Oct. 3, 1927. She had had attacks of pain, vomiting, and jaundice ever since she could remember—certainly since she was a small child.

Pain was felt right across the upper abdomen, in the lower chest, and in the back, but not in either shoulder. It came on in attacks of severity varying from slight to severe, and lasting from a few hours to a day or so. Nothing, dietary or other, had been noted to precipitate the attacks, which came on at any time of the day or night and had increased in frequency latterly. Often the pain persisted as a dull one having the same distribution as in the attacks, and for some months she had hardly been free from it.

Vomiting always accompanied the painful attacks and was incessant while they lasted. Nothing appeared to control it, although at times it seemed to relax the severity of the pain.

Jaundice always followed the attacks at something less than a day's interval; slight at the onset, it at times became deep, when it was usually accompanied by itching of the skin. She had been seen by several observers on different occasions since May, 1927, and though on none of these had she been deeply jaundiced, only once had she been free from a tinge.

Her *appetite* for all foods had always been bad.

Constipation had always been present, and she had difficulty in opening her bowels with purgatives. The stools were pale and the urine was dark when the jaundice was severe.

She had never noticed any swelling of the abdomen, but during the attacks the upper abdomen was tender. She was first observed by the writer a few days after one of the attacks.

ON EXAMINATION.—She was seen to be thin, ill, and lethargic. She was slightly jaundiced in the skin, mucous membranes, and conjunctivæ. The tongue was clean. In the abdomen a vague mass could be felt below the right costal margin, immobile with respiration, but too indefinite to permit the recognition of any special characters other than that it seemed to be

deeply placed and that the abdomen over it was resonant to percussion. The liver was not felt. Her temperature was normal; the pulse 80 and of poor volume; blood-pressure 116.

A diagnosis of cystic dilatation of the common bile-duct was suggested, and the following examinations were made: Cholecystography (after oral administration of 6 grm. of tetraphenolsulphonaphthalein): no gall-bladder shadow was visible, but part of the drug was evident in the colon unabsorbed.

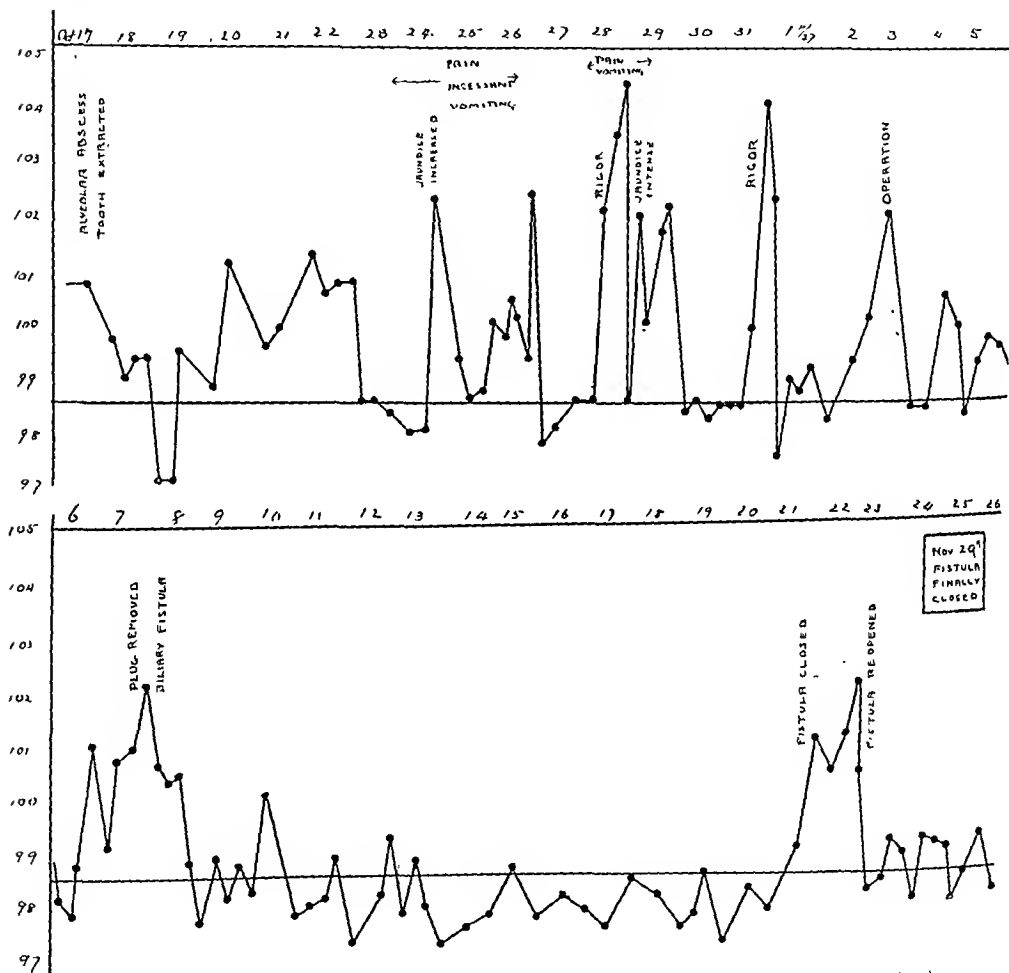


FIG. 283.—Case of N.P. Temperature chart. (Reproduced from Author's drawing.)

Corpuscular fragility was found to be within normal limits. Red and white blood-cell counts were normal. Van den Bergh's reaction was directly biphasic, indirectly positive.

She was admitted to University College Hospital on Oct. 19, 1927, when she was found to be suffering from an alveolar abscess that was associated with pain, swelling, and pyrexia amounting to 101.6°. It was necessary to extract an upper molar tooth, after which her jaw cleared up slowly.

but, as is seen from her chart (*Fig. 283*) the fever did not. Apart from her dental condition, no physical signs of disease were now appreciable, other than a very slight tinge of jaundice. There was no abdominal swelling.

On the night of Oct. 23 she had an attack like those described in her medical history. The pain was felt in the upper abdomen, lower chest, and back, but not in either shoulder or scapular region. It was severe, and she was given morphia. There was incessant bilious vomiting and retching. On examination of the abdomen there was now an indefinite globular mass palpable in the right hypochondrium, crossing the middle line and extending for $2\frac{1}{2}$ in. below the right costal margin. It was tender, immobile with respiration, and resonant on percussion. There was also diffuse tenderness over the upper half of the abdomen. On Oct. 24 the jaundice had increased and the temperature rose to 102.2° . The abdominal mass, which was still tender, was now a little more definite and was thought to be about the size of a lawn-tennis ball. The vomiting continued for two days and the jaundice persisted.

On Oct. 28 there was another attack, this time associated with a temperature of 104.4° and a rigor, the jaundice subsequently becoming intense, the stools clay-coloured, and the urine dark with bile. The pulse frequency now rose to 160, and did not fall below 120 until after her operation. A blood-count now showed a leucocytosis of 11,500 per c.mm., and of these 74 per cent were polymorphonuclear.

Laparotomy had been decided upon, but, owing to the supervention of what was clearly an acute infective cholangitis, was postponed as it was thought desirable that the infective condition should subside if it would before operation. On Nov. 1, however, she had another rigor with further high fever and pulse of 150, and she appeared to be going downhill. Operation was therefore deferred no longer.

Since her admission to hospital calcium chloride had been administered by the mouth, because it was thought, in view of the known danger from hæmorrhage in these cases, that no pre-operative precaution should be omitted, however doubtful its value.

OPERATION.—On Nov. 3, her temperature at that time being 102° and the pulse 120, the abdomen was opened under ether by a right paramedian incision. No adhesions were found, but the gall-bladder was seen to be enlarged, flattened, slackly distended, and thickened, being $4\frac{1}{2}$ in. long and $2\frac{1}{2}$ in. broad in its widest part. On displacing the gall-bladder and liver upwards, a globular distention of the common bile-duct was seen, about the size of a Tangerine orange, extending upwards as far as the entrance of the cystic duct, and downwards behind the second part of the duodenum. The cystic duct was short, wide, and straight, and apparently entered the upper part of the cyst of the bile-duct. On reflecting the peritoneum covering the cyst, hæmorrhage from a network of thin-walled and distended veins occurred, and on an attempt to isolate the cyst from its surroundings the bleeding became free and difficult to stop except temporarily by gauze pressure. The wall of the cyst was friable and did not come easily away from surrounding structures. A similar experience was met with in attempting to define

accurately the junction of the cystic duct with the cyst. The accompanying diagram (Fig. 284) shows the operative findings, but the exact nature of the distention, whether fusiform, lateral, or saccular, was not ascertained for the reason given, nor was the possible presence of stricture of the bile papilla investigated.

In view of the small size of the cyst and of the troublesome bleeding attending its slightest manipulation, and in view of the evil condition of the

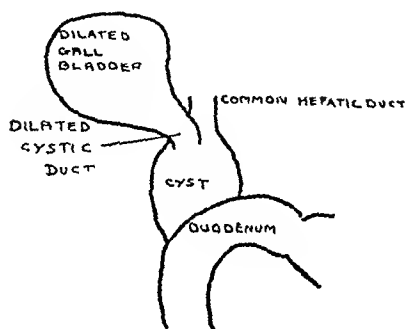


FIG. 284.—Condition found at operation.
(Reproduced from Author's diagram.)

patient resulting from biliary infection, it was decided to join the gall-bladder to the stomach, a procedure which would be quick and easy, and need not involve bloodshed. A plug had been placed over the cyst for hæmostasis, and after completion of the anastomosis this was removed, but considerable bleeding persisted from a large vein near the junction of the cyst with the cystic duct. In an unsuccessful attempt to catch the vessel with forceps the cystic duct was punctured. This opening was closed with fine catgut. A plug was placed on the bleeding

vessel and a tube in the wound, which was then closed with catgut. A culture taken from bile aspirated from the cavity of the gall-bladder yielded a pure growth of *Bacillus coli*.

The patient stood the operation well, and by the following day the itching that had been present was relieved. The temperature came slowly and irregularly down during the succeeding days. The plug was taken out on the fourth day, removal being followed by a free discharge of bile.

The jaundice cleared up slowly but completely during the ensuing fortnight, and bile gradually returned to the stools. The biliary fistula persisted until Nov. 21, when its closure resulted in an attack of slight pain and a rise of temperature to 102°. Another copious discharge of bile was followed by gradual subsidence of the fever. The fistula finally closed on Nov. 29, and the patient left hospital healed on Dec. 7.

AFTER-HISTORY.—Three and a half months later (March, 1928) she was well, all symptoms having disappeared. There was no trace of jaundice, she had a good appetite for the first time in her life, could 'eat anything', and, having been previously thin, was now noticeably fatter. Constipation had completely disappeared and she no longer took purgatives. As an individual she was brighter, and the lethargic mentality previously obvious was not now discernible.

COMMENTS.

In view of the large number of writings dealing with this malady that have appeared in recent years, and in particular in view of that of Professor Choyce, so far unpublished but referred to here in the opening paragraph, the writer refrains from discussing the disease at length, but the following observations may be permitted.

Diagnosis.—Neugebauer's case was that of a woman of 21 with seven months' history of abdominal pain and one month's of abdominal swelling and jaundice. She had a cyst in the abdomen that was easily palpable as a large fluctuating swelling that proved at operation to contain $3\frac{1}{2}$ litres of dark fluid. The enormous size to which the cyst had grown was probably responsible for the ill fortune of the event.

In the present example the abdominal swelling was always indefinite, and, though undoubtedly palpable, its nature could not have been recognized without the presence of other features of the disease. The development of a tumour while an attack was under observation clinched a diagnosis already the one most probable. The writer would thus emphasize the clinical history as the essence of the diagnosis of this malady, and to this end he has described at some length that of one case. The essential clinical features appear to be those of attacks of abdominal pain felt also in the back but not in either shoulder, accompanied by continual vomiting and followed by jaundice. If occurring in young women or if dating from early childhood, such attacks should suggest the disease in question as their cause.

Infection.—While this patient was under observation there occurred serious infection, so that her life was jeopardized. It is possible that cases arise where infective cholangitis with cholecystitis are recognized and treated by drainage of the gall-bladder, the underlying cause of cystic dilatation of the common bile-duct escaping observation owing to the dilatation of the gall-bladder that usually accompanies the condition, and to the necessity for the adoption of a quick and simple procedure in patients seriously ill. This might easily have occurred in the present example had the patient not been seen previously both in a quiescent period and when suffering from biliary obstruction without infection.

Operative Treatment.—In this girl the distention of the common bile-duct was moderate and could be recognized; but an accurate dissection, showing exactly the type of dilatation, whether fusiform, globular, or saccular, and investigating the condition of the bile papilla, was impossible, because an attempt resulted in free bleeding, an increase of which was unjustifiable on account of the precarious condition of the patient arising from infection. It is likely that a part of the bleeding was due to the infected state of the wall of the cyst, which was adherent to surrounding structures and was unpleasantly friable. The real difficulty of hæmorrhage thus made it unjustifiable to attempt anything but the easy anastomosis of the gall-bladder to the stomach. The subsequent history shows that in this girl's case at any rate, where the cyst was small, cholecystgastrostomy was adequate. Hæmorrhage has accounted for a proportion of the deaths that have followed operations for this malady, and the writer is of opinion that once it has been recognized, either before or at operation, where the cyst is small, anastomosis of the gall-bladder to the stomach or duodenum is the proper treatment, because with such a procedure there can be no danger whatever of bleeding from the veins around the cyst, and because it has certainly given this patient complete relief. The operation widely advocated for cyst of the common bile-duct is that of anastomosis of the cyst to the duodenum. But where a cyst exists

the gall-bladder is as a rule dilated, and it is possibly better to short-circuit the bile from its first distended reservoir, as it may be that a recurrent residual cholecystitis will arise later where the second distended reservoir, the cystic common duct, has been drained into the alimentary canal and the gall-bladder left alone.

REFERENCES.

- ¹ JUDD, STARR, and GREENE, EARLE, *Surg. Gynecol. and Obst.*, 1928, xlv, 317.
² NEUGEBAUER, F., *Beitr. z. klin. Chir.*, 1924, cxxxi, 448.

A CASE OF NON-ROTATION OF THE MID-GUT.

By J. W. THOMSON,

HONORARY SURGEON TO THE CLAYTON HOSPITAL, WAKEFIELD.

DERANGEMENTS of the second stage of intestinal rotation are divided, as described by Norman Dott in the *BRITISH JOURNAL OF SURGERY*, vol. xi, p. 266, into three groups: (1) Non-rotation of the mid-gut loop; (2) Reversed

rotation of the mid-gut loop; (3) Mal-rotation of the mid-gut loop. I have to record a case of the first group.

D. G., female, age 23, was admitted to the Wakefield Hospital on June 30, 1927.

HISTORY.—The patient was quite well until the age of 18, when symptoms referable to the digestive organs set in. Her condition gradually deteriorated, pain after food increased in severity, and vomiting became more frequent till only small quantities of food could be retained. Prolonged medical treatment had given no relief.

CONDITION FOUND AT OPERATION.—The first part of the duodenum passed somewhat vertically upwards, and the second part passed downwards almost parallel with the first, forming an acute angulation at their junction. Slight traction on the second part brought it into immediate contact with the first part. The duodenum then



FIG. 285.—The duodenum is seen passing to the right, and the jejunum and ileum are occupying the right hypochondriac, right lumbar, and right iliac regions.

became freely movable and passed outwards to the right to join the jejunum. The jejunum, with the ileum, occupied the right hypochondriac, right lumbar,

and right iliac regions. The positions of the duodenum, jejunum, and ileum are well shown in *Fig. 285*. The colon presented a striking departure from its normal formation. At its centre the transverse colon descended into the pelvis in the shape of a narrow V. Slight traction on the apex of the V brought the sides into close contact, and when this loop was spread out, its upper extremities were not more than $1\frac{1}{2}$ in. apart. From those points the colon passed horizontally outwards to the splenic and hepatic flexures. The small intestine was now displaced to the left, and this brought into view the cæcum, which was reversed, with the appendix on its upper aspect. The ileum entered the cæcum from the right side. The ascending colon was absent from its usual site. It was deeply incurved, reaching at least to the middle line under cover of the dependent loop of transverse colon.

OPERATION.—The duodenum just beyond the pylorus was anastomosed with the distal extremity of the second part, so as to get rid of the angulation at their junction. The dependent loop of transverse colon invited resection, but I chose the alternative of colocolostomy at the upper end of the loop. The appendix was removed, and the cæcum and ascending colon were fixed to the posterior abdominal wall by the method of Waugh. This is seen in *Fig. 236*.



FIG. 286.—Shows the cæcum and ascending colon now fixed to the right posterior abdominal wall, and also the site of the colocolostomy.

The combined operations of duodenoduodenostomy, colocolostomy, appendicectomy, and cæcocolopexy have resulted in the patient's complete recovery.

ENTERIC CYST OF LARGE SIZE IN A BOY.

By E. G. SLESINGER,

ASSISTANT SURGEON TO GUY'S HOSPITAL, LONDON.

L. M., MALE, age 7 years, was brought with a history of 'stomach-ache' during the last three or four years. This consisted of attacks of generalized abdominal pain of a gripping character, associated with vomiting. Each attack had been treated by rest in bed and starvation and had cleared up in three or four days. Intervals of six months with complete freedom from symptoms had occurred

between the attacks. There had been no change noted in the motions, no constipation, and no disturbance of micturition. On examination of the abdomen a tumour was found to be present slightly to the left of the mid-line and below the umbilicus. It was rounded, freely movable, rather bigger than a large orange, not tender. Rectal examination revealed no abnormality.

OPERATION.—The abdomen was opened, and the tumour, which was bluish-black in colour, was seen to be a cyst attached to the small intestine

at a point approximately 8 feet from the ileocaecal valve. It was removed, together with some inches of the small intestine to which it was attached (*Fig. 287*), and the continuity of the gut was restored by end-to-end anastomosis. It was noted that the mesentery of the small intestine from the spine to the attachment of the cyst showed bands of condensation and thickening on its surface, similar to those seen in association with severe constipation. The patient made an uneventful recovery.

DESCRIPTION OF THE SPECIMEN.—On opening the cyst it was found to contain masses of mucus in a blood-



Fig. 287.—Appearance of tumour after removal.

stained fluid. There was a complete septum between the lumen of the intestine and of the cyst, but the intestinal wall could be seen to be continuous with that of the cyst. There was a small papillary growth on the mesentery at its junction with the bowel opposite to the cyst.

Microscopic examination of sections of the cyst wall by Dr. G. W. Nicholson showed two layers of muscle, and a lining of fibrous tissue with no demonstrable epithelium. Section of the papilloma showed it to consist of dilated lymphatics with some muscle tissue.

Comments.—The nature of this cyst is obscure, and in the absence of an epithelial lining it appears impossible to be certain of its origin. Its position at 8 feet from the ileocaecal valve makes its origin from Meckel's diverticulum improbable—the complete covering of the cyst with two layers of muscle is unlike the usual structure of intestinal diverticula. The possibility of its origin as a distention cyst of a mucous gland cannot be excluded in the absence of any epithelial lining, though both its size and its position are against this view; but in many respects such an origin appears to be the only alternative to the assumption that it is a Meckel's cyst in an unusual position.

**VOLKMANN'S ISCHÆMIC CONTRACTURE TREATED BY
TRANSPLANTATION OF THE INTERNAL EPICONDYLE.**

By HAMILTON BAILEY,

SURGEON TO THE DUDLEY ROAD HOSPITAL, BIRMINGHAM.

ON July 10, 1927, J. F., age 4, whilst attempting to jump over a tennis net, caught his foot in the webbing, and fell. He was taken forthwith to hospital, where it was found that he had sustained a transverse fracture of the lower end of the humerus. The fracture was treated in the casualty department, the arm being put up in flexion and supination and the position maintained by means of adhesive strapping and bandaging. Late on the same evening

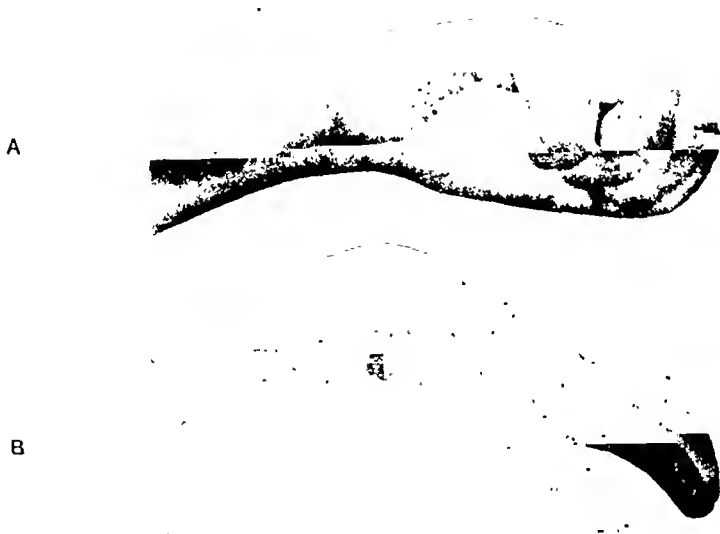


FIG. 288.—Photograph showing: A. The Volkmann's contracture; B, That the fingers could be partially extended by flexing the wrist.

the mother noticed that the child's hand was blue and swollen, and she again brought the child to the casualty department, where the strapping and bandage were loosened. On July 24 he attended the massage department and, to quote the report received from the massage sister, "he was given the usual massage treatment, and after one week we noticed what appeared to be an ischæmic contraction."

On July 29 the child was brought to my out-patients' department. On examination it was apparent that he had a well-marked Volkmann's contracture. The photographs (Fig. 288, A, B), which were taken a few days later, illustrate the fact that the fingers could only be straightened when the wrist was acutely flexed. A skiagram of the elbow-joint showed that the

fracture had united in very good position. The patient was admitted. Postural treatment by flexion of the wrist over a moulded plaster splint, combined with massage and movement, was persevered with. During this time the hand was manipulated on two occasions under a general anæsthetic, but the flexor tendons were obviously permanently shortened, and resisted stretching like miniature hawsers.

Although the massage staff worked diligently on the case, by the end of October there was no sign of any improvement, and the parents were naturally agitated at the lack of progress and the possibility of a permanent

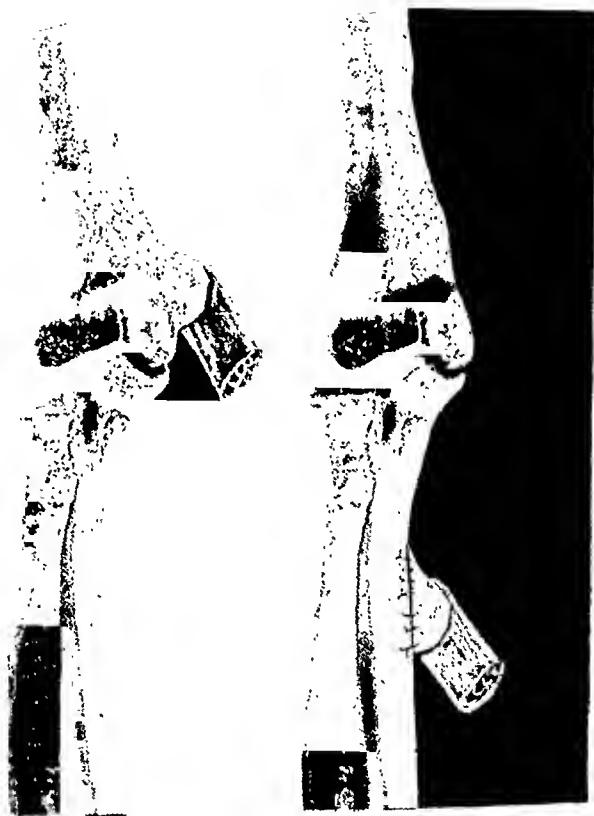


FIG. 289.—Demonstrating the principle of the method described. The internal epicondyle with its common flexor origin still intact is transplanted into the shaft of the ulna.

deformity. This was the determining factor in deciding that a more active course should now be taken.

OPERATION.—On Nov. 6, 1927, the following operation was performed, which is a slight modification of the method originated and described by Max Page.¹

An incision was made on the inner side of the elbow, and the ulnar nerve isolated and hooked out of harm's way. The bicipital fascia was divided, and the origin of the flexor muscles carefully dissected from the upper third

of the ulna, but the main common origin from the internal epicondyle was left intact. When this dissection was finished, a large mass of muscle under which the finger could be passed was attached above to the internal epicondyle. The condyle was then cut off and sutured into a little bed made by reflecting up the periosteum on the inner side of the shaft of the ulna at the junction of its upper third to the lower two-thirds (*Fig. 289*).

RESULTS.—The immediate result of the operation confirmed Max Page's observation that there is no sudden relief of the contracture. After fourteen days the fingers began to straighten, and by the end of the third week they could be completely extended. Massage and movement were continued for eight weeks. Seven months later (June, 1928) the patient has almost perfect

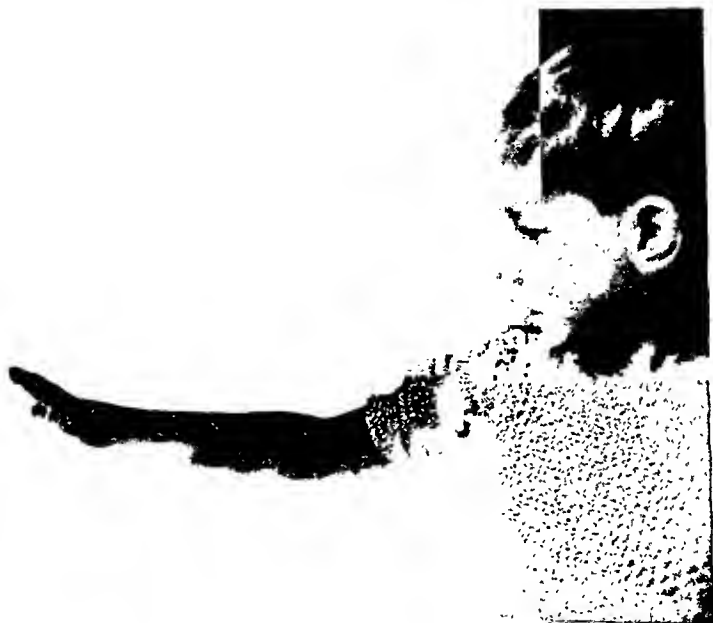


FIG. 290.—Six months after the operation: showing that the fingers can be completely straightened.

function in the limb. His mother states that he uses his hand normally in every way. *Fig. 290* shows that the fingers can be completely straightened, but on examination there is still some slight resistance to hyperextension of the wrist.

No definite conclusion can be drawn from the treatment of one case by a particular method; but there can be no doubt that in this instance the operation rectified in the span of a few weeks a contracture which had remained stationary after four months' persistent conservative treatment.

REFERENCE.

- ¹ PAGE, MAX, *Jour. Bone and Joint Surg.*, 1923. April, 233.

INTERARTICULAR DISLOCATION OF THE PATELLA.

By J. PENRY JONES,

SENIOR HOUSE SURGEON TO THE RADCLIFFE INFIRMARY, OXFORD.

E. I. B., age 11, was admitted to the Radcliffe Infirmary, on November 27, having injured her right knee about two hours previously, through slipping on the edge of a pavement and over a bicycle in the dark. On examination there was already a marked effusion into the joint, but no external appearance of injury; extension was limited to 150° , but flexion was almost normal. The patella seemed smaller than usual, but no fracture could be made out; above the patella was a marked hollow, the quadriceps extensor expansion on each side of this being drawn tight. A radiogram showed that the patella had rotated backwards about a horizontal axis through 90° , so that



FIG. 291.—Skiagram showing impaction of patella and also the small piece of ossifying cartilage (α) still attached to the posterior aspect of the extensor expansion.



FIG. 292.—Condition of the knee seven weeks after operation.

its upper end was engaged in the intercondylar notch of the femur. The limb was prepared for open operation.

Manipulation under an anæsthetic next morning failed to reduce the dislocation. The joint was therefore opened by an external semilunar incision, the capsule being slit vertically just to the outer side of the patella. After releasing about six ounces of blood-stained effusion the upper end of the patella could be seen wedged in the intercondylar notch; the quadriceps extensor insertion had been stripped clean off the upper and anterior surfaces of the bone, and its posterior surface still carried a small piece of ossifying cartilage from the upper end of the patella (Fig. 291). The bone had to be

forcibly levered before it could be disengaged, when it immediately assumed its normal position; on extending the joint it could be seen that it was not necessary to do more than suture the slit capsule and skin. A plaster splint was kept on for about four weeks, only being taken off for massage and movements. The patient made an uninterrupted recovery, and seven weeks after the accident (*Fig. 292*) the range of movement at the joint was normal.

Comments.—The mechanism of this injury, of which there are to my knowledge only fourteen examples in the literature, has never been satisfactorily explained. Scott insists on two separate impacts to explain its occurrence, and both his cases give that history. Paterson Brown's case had an open wound due to a 'glancing blow', and it was uncertain whether there was a direct blow or not. The inevitable absence of a definite account of the mode of injury makes it impossible to dogmatize on the mode of occurrence, but it may be that the first movement is an extreme and forcible flexion of the knee, which leaves the upper end of the patella on a level with the intercondylar notch of the femur; this is rapidly followed by extension; the patella remains in its newly adopted position, and the quadriceps, stripped off the anterior surface of the patella, now acts on the lower border, thus wedging it firmly between the two condyles of the femur. A point of interest in this particular case, and one that may have some bearing on the etiology of the condition, is the fact that the patient was 'double-jointed' in most of her joints; the middle finger, for instance, could be bent backwards to touch the back of the wrist with incredible ease.

I have to thank Mr. Hugh Whitelocke for permission to publish this case, and also Dr. Sankey for his excellent radiograph.

BIBLIOGRAPHY.

- ANA, G., "Downward Dislocation of the Patella", *Jour. Amer. Med. Assoc.*, 1922, lxxvii, 1457.
 BROWN, K. PATERSON, "Interarticular Dislocation of Patella", *Edin. Med. Jour.*, 1924, July, 403.
 COOPER, ASTLEY, *A Treatise on Dislocations and on Fractures of the Joints*, London, 1823, 183.
 DEMARQUAY, "Mémoire sur les Ruptures du Tendon du Triceps fémoral, au-dessus de la Rotule." *Gaz. méd.*, Paris, 1842, x, 593.
 HAMILTON, F. H., *A Practical Treatise on Fractures and Dislocations*, London, 1891, 760.
 NEWMAN and RUTHERFORD, H., "Two cases of Dislocation of the Patella", *Lancet*, 1901, i, 1761.
 PERKINS, J. W., "Hitherto Undescribed Dislocation of the Patella Endwise", *Jour. Amer. Med. Assoc.*, 1920, lxxiv, 388.
 RUTHERFORD, H., "Downward Dislocation of the Patella", *Brit. Jour. Surg.*, 1921, viii, 524.
 SCOTT, C. M., "Two Rare Cases of Dislocated Patella", *Jour. Amer. Med. Assoc.*, 1924, lxxvii, 1198.
 SZYMAN, L., "Ueber eine seltene Form von Patellar-Luxation", *Deut. Gesellschaft f. Chir.*, 1889, 259.

AN UNUSUAL CASE OF INTESTINAL OBSTRUCTION.

BY LIEUT.-COLONEL H. HALLILAY, I.M.S., RAWALPINDI.

THE following case appears to be worthy of recording on account of its rarity.

HISTORY.—Isher Singh, male, age 40, Hindu, admitted in August, 1926, for pain in the right side and a gradually increasing constipation, gave the following history. About sixteen years ago, after one of the Hindu feasts, he was returning to his home after indulging in some country liquor, when he suffered an attack of pain in his right side. This recurred at intervals for some years afterwards, the pain being always in the right iliac fossa and accompanied by fever. Of late these attacks had recurred, but without fever. For the last two months the pain had been almost continuous and his life rendered intolerable by it.

ON EXAMINATION.—There was a lump in the right iliac fossa, and the movement of distended coils of intestine were distinctly visible. There was no tenderness in the right iliac fossa.

OPERATION.—The abdomen was opened in the middle line, the rectus being displaced outward. The lower two feet or so of the ileum were involved in a nest of adhesions, as was the commencement of the cæcum. A large, firm, rounded body was found in the ileum just at the junction of the bowel with the cæcum. It was found to be impossible to separate the adhesions, which were very firm and abundant. The bowel was incised over the most prominent part of the swelling, and a large enterolith was removed from the ileum. The stone and the ileum had formed a perfect ball-and-socket valve which at times sealed up the opening of the ileocæcal valve and prevented the passage of the ileal effluent into the cæcum. The muscular coats of the ileum were enormously hypertrophied. The incision in the bowel was closed, and the lower end of the ileum was short-circuited with the colon. The man's symptoms were all cleared up by the operation and he made an uneventful recovery.

Comments.—It is probable that the enterolith was formed in the tortuous and stagnating passages of the last two feet of the ileum after the appearance of the adhesions. The adhesions must have been the sequel of the several attacks of appendicitis of which he gave the history. The alternative hypothesis is that the first attack of pain was caused by the impaction of the stone at the entrance of the ileocæcal valve, and that the subsequent attacks were due to the recurrence of partial obstruction from the same cause. In view of the rarity of cases of appendicitis amongst the country-bred Panjabi, this is worth considering.

REVIEWS AND NOTICES OF BOOKS.

The Abdominal Surgery of Children. By L. E. BARRINGTON-WARD, Ch.M., F.R.C.S., Surgeon to the Hospital for Sick Children, Great Ormond Street, and to the Royal Northern Hospital, London. Demy 8vo. Pp. 283 + xiii, illustrated in colour and in black and white. 1928. London: Humphrey Milford. 15s. net.

At first sight it would seem that this volume might be regarded as a stick to be put into the hands of those who are inclined to belabour the modern tendency to increasing specialism in surgical work. Here is a volume of some 270 pages designed with due deliberation to present a special portion of what has come to be regarded as a specialty, the surgical work of infancy and childhood. It might appear that this is an example of the objection which the repudiators of ultra-specialism so strongly decry, the principle of *divide et divide*. The volume, however, permits of no reasonable objection on that score. Mr. Ward, from a wide experience in general surgery, has expressed his ideas and views upon the various abdominal ailments which are either peculiar to, or are reasonably common to, the periods of infancy and childhood. There is therefore no justifiable ground for criticism of the conception, for the idea is really an excellent one, and, if any further excuse is demanded, it may be found in the fact that many of the most distinctive features of pædiatric surgery are concerned with abdominal disorders.

The first chapter is in some ways the most interesting and suggestive contribution to the volume; it is concerned with the general principles of the operative treatment of the abdominal derangements. It is replete with valuable information. It may be described as an apt presentation of the teaching of the Great Ormond Street School, and if certain of the views expressed are open to criticism it is none the less a helpful and erudite production. The complication of ketosis is given full and detailed consideration; more might have been made of the diagnosis of the condition, particularly of the distinction between ketosis and appendicitis. For example, no mention is made of the value of the leucocyte count as a means of differential diagnosis. On the other hand, it would seem that undue importance is attached to the risk of confusing ketosis with acute volvulus of the small intestine. It is true that Le Wald's investigation indicated a laxity of the ileocecal attachment as being common to many cases which demonstrate cyclic vomiting, but he read his findings as indicating a constitutional error of the involuntary nervous system rather than a local error producing a local effect.

A well-reasoned account is given of the surgery of the abdominal wall, but in terms of anatomy is it correct to speak of supra- and infra-umbilical glands? Are there such structures? We do not agree with the statement made under "Umbilical Hernia" (p. 27) that there is "a separation of the normal muscles".

Hernia is classed under abdominal conditions, and full consideration is given to the various aspects of this common complaint. In describing the descent of the testis the statement is made that "as the testis descends into the scrotum it invaginates the processus vaginalis". The majority of embryologists would contest this statement, and the late Dr. Berry Hart contradicted the view on more than one occasion. The statement ignores the fact that, while the processus vaginalis is lined by endothelium (mesothelium), the covering of the testis is germinal epithelium.

In this chapter, as in other parts of the volume, Mr. Ward employs some unusual metaphors and idioms—he speaks of 'public school games', of operations postponed until the child is 'definitely established', and of a hernia sac being opened

'to ensure that it contains nothing'. It may be considered out of place to criticize syntax in the review of a scientific publication, but there can be no doubt that medical literature would benefit if more attention were paid to the principles of grammar and euphony, and this volume is no exception to the rule.

The chapter dealing with the surgery of the stomach and duodenum is an excellent contribution. The figures and facts relative to congenital stenosis of the pylorus are perhaps the most striking endorsement we have read of the merits of operative interference in contrast to medical treatment; the statistics quoted on p. 85 and the succeeding pages are a testimony which cannot be ignored.

A curious and interesting observation is made on p. 103 in relation to the post-operative complication of hyperpyrexia. After drawing attention to the seriousness of the complication, the statement is made, "As a post-operative complication it is now rare, although in the years 1918-20 it was comparatively frequent". We wish Mr. Ward had supplied the key to this intriguing problem. As it is, he has left us tantalized and 'in the air'—an exercise of the author's prerogative, perhaps!

Mr. Ward's work on Hirschsprung's disease is widely known, and his further contribution to the problem is of outstanding merit, but he might have included with advantage some reference to the striking results which Wade and Royle have obtained by the operation of ramisection in their cases as recorded in the *Medical Journal of Australia*, January, 1927.

Speaking generally, the volume is a worthy contribution to medical literature. The illustrations are numerous and beautifully reproduced. In future editions the merits of the work will be enhanced if fuller references are given to the recent and current literature of the various subjects.

A Manual of Proctology. By T. CHITTENDEN HILL, Ph.B., M.D., F.A.C.S. Second edition. Demy 8vo. Pp. 294 + vi, with 101 illustrations. 1926. Philadelphia and New York: Lea & Febiger. \$3.50.

THIS concisely written book will be found to be a very useful guide to those who desire to possess a sound practical knowledge of proctology. The opening chapter deals with methods of examination and diagnosis, and contains some valuable hints for conducting an investigation with the sigmoidoscope. The three succeeding chapters are concerned with the inflammatory and ulcerative conditions of the rectum and with ulcerative colitis. In the treatment of these morbid states great emphasis is laid upon the value of solutions of nitrate of silver, with which we entirely agree; but we are surprised to find that treatment by an autogenous vaccine, which is often very efficacious in these cases, is not mentioned.

A succinct account of anal fissure is to be found in Chapter 5. When discussing etiology the author favours the view that the formation of a fissure is in most cases due to the fact that the mucous membrane at the posterior commissure of the anus is insufficiently supported by reason of the anatomical arrangement of the fibres of the external sphincter muscle. We do not concur with this explanation, because if a fissure were due to anatomical defect the lesion would be more common than it is. The fact that a fissure is often permanently cured by the simple procedure of dividing the external sphincter muscle is a sound argument against anatomical arrangement of the muscles being an etiological factor. We agree with the author in the opinion that the operation of incision gives more satisfactory and more permanent results than can be obtained either by excision of the fissure or by forcible dilatation of the sphincters.

Rectal abscesses are described in Chapter 6, the anatomical situation of the varieties being well depicted in clear diagrams. The pelvirectal variety is especially well described. In the surgical treatment of this form of abscess the author emphasizes the advisability of dividing the levatores ani transversely on both sides so as to ensure adequate gaping of the wound. We agree with this to a certain extent, but would point out that if the levatores are divided too extensively the power of expelling the rectal contents may be seriously interfered with.

The next chapter is devoted to the consideration of the important subject of fistula. The usual classification into complete, blind internal, and blind external fistulae is adhered to. Our objection to this classification is that it presumes a single type. The failure to appreciate the fact that there are four distinct types of ano-rectal fistulae, each presenting the three varieties mentioned, is, we think, responsible for some of the disastrous consequences of operative interference. On page 106 the author makes this statement: "If the opening does occur above the internal sphincter, and provided that it is posterior to the rectum, it is my practice (although I am aware that there are many high authorities who do not believe in ever dividing the internal sphincter) to pass the director into the rectum and divide all the intervening tissue, which necessarily includes the sphincters, both external and internal. In my experience the wound heals rapidly and there is very little danger of incontinence." We regard this as very dangerous teaching. If the whole of the tissues between the external and internal openings of a fistula are divided, the risk of resulting incontinence is entirely dependent upon the anatomical situation of the main track of the fistula. When the main track is situated internally to the muscular coat of the bowel no harm results; but if it is situated outside the rectal wall grave incontinence always ensues. It is probable, therefore, that the cases of fistula with high-lying internal openings that have been treated by the author in this manner have been those whose main tracks have been confined to the subcutaneous and submucous tissues. This seems likely, because, after concurring with the view that 90 per cent of fistulous tracks are placed superficially to the external sphincter, he remarks on page 102: "Even when the probe seems to pass rather deeply, and one would be under the impression that he was dividing the external sphincter muscle, if the incision is made with a scalpel cutting down on the probe, it is found that no muscle fibres are divided." Apart from this the subject of fistula has been dealt with exceedingly well, and some excellent rules to be observed during both the operative and the after-treatment of fistulae have been ably expounded. The diagrams representing the various kinds of fistulae are clear, but the explanatory note under the letter F in Figs. 28, 33, 34, and 35 is obviously wrong.

Chapter 8 is concerned with stricture of the rectum. We agree that a very small percentage of strictures of the rectum are of syphilitic origin. When discussing the treatment of annular strictures, linear proctotomy is recommended, and we are warned against dividing the internal sphincter lest incontinence should follow. This warning is perhaps a little curious, because the author has told us that he does not hesitate to divide that muscle in certain forms of fistula. Excision of the rectum for extensive tubular stricture is rightly condemned.

The subject of hæmorrhoids is very fully considered, and the various operative procedures that are usually adopted are described in detail. The author expresses his predilection for local anaesthesia, and carefully describes his technique.

The classification of prolapse of the rectum into prolapse of the mucous coat only and complete prolapse of all the coats will meet with general approval, but we cannot agree with the writer when, speaking of the operative treatment of complete prolapse, he says: "I feel very strongly that it should never be employed in the ordinary run of cases, as the operation is more or less mutilating to the lower musculature of the rectum: the levator ani must be divided: the internal sphincter is sacrificed, though the external sphincter may be saved, and it is impossible to attach them to the amputated gut in such a manner that they will ever function normally again." The whole point of the operation is that the incision through the wall of the rectum should be made about an inch from the mucocutaneous junction in order to avoid injury to the levatores and sphincters. It has been our experience that excellent control over the rectal contents is maintained after the operation.

Pruritus ani, rectal incontinence, and benign tumours are cursorily considered in the following chapters, and the final chapter is contributed by Dr. Coffey, who gives his reasons for performing the radical abdomino-perineal operation for cancer of the rectum in two stages.

We have read this book with great pleasure, and consider it to be a very sound guide in the treatment of rectal diseases.

Gonococcal Infection in the Male. By ABR. L. WOLBARST, M.D.; with a chapter written by J. E. R. McDONAGH, F.R.C.S. Medium 8vo. Pp. 237, with 89 illustrations including 7 colour plates. 1927. London: Henry Kimpton. 25s. net.

THE author of this monograph is well known as a great authority on venereal diseases; perhaps he is best known in England as the inventor of the 'five-glass test' in the diagnosis of urethral discharges in the male; this test has always seemed to us to be the most scientific and the most satisfactory of the many tests which have been devised.

He has written a book on a subject in which he is intensely interested, and of which he has had an immense experience, and the result is naturally excellent; he gives a clear and convincing account of the pathology, diagnosis, and complications of gonococcal infection, and special sections on subjects of great importance, such as 'Diathermy in Gonorrhœa', 'When is Gonorrhœa cured?' 'Sexual Neuroses and Male Sterility following Gonorrhœa', and 'Personal Prophylaxis'.

We consider the chapters on treatment to be the least satisfactory in the book; this is not the fault of the author, who gives a full account of the numerous remedies that may be employed; it would rather seem to be due to the highly refractory nature of the organism. It really appears as though there has been very little progress in this respect; Janet's method of irrigation is, the author states, falling into disfavour, and syringe injections are replacing this—only, instead of using potassium permanganate, various new silver salts have taken its place.

The author writes: "There is no short cut to success in the treatment of acute gonorrhœa; avoid complications, and do not worry about the time it takes to bring about a cure": this rather suggests that the patient cures himself by some process of acquiring immunity.

Dr. Wolbarst is much interested in the newer methods of treating the complications, such as diathermy, intravenous injections of various drugs, autoserotherapy, and protein therapy; from all of these he seems to have had most satisfactory results, and the reader is left a little bewildered as to which of these he is to use or whether he is to use them all.

In the chapter on chronic seminal vesiculitis, the author states that Belfield's operation of vasotomy, with the injection of an antiseptic fluid into the vesiculæ by this route, has proved "of inestimable value". We cannot see that he brings forward any proof that the fluid ever enters the vesiculæ; moreover, he adds that the fluid thus injected can be recovered almost immediately through a catheter inserted in the bladder; unless there is some obstruction in the ejaculatory duct, we see no reason why the fluid should not flow straight through into the prostatic urethra.

We should like to enter a mild protest against Dr. Wolbarst's fondness for placing the adverb before the verb; in one instance he says, "fluctuation always cannot be felt, because the pus-area may be deep seated"; the meaning would be much clearer if he had said, "fluctuation cannot always be felt"; in still another instance he writes: "those provoking cases in which post-nuptial coitus with a virgin rapidly is followed by an acute outbreak of gonorrhœa"; here the reader is frankly puzzled as to whether the rapidity is to be construed as qualifying the coitus or its unfortunate consequences. These, of course, are minor defects, and do not alter the fact that Dr. Wolbarst has produced a very fine monograph on gonorrhœa in the male.

In the chapter by Mr. McDonagh we find an account of "his unique views on gonorrhœa"; the words are not ours but Dr. Wolbarst's.

Was kann und wann muss der praktische Arzt operieren? By Professor R. MÜHSAM, Director of the Rudolf Virchow Hospital in Berlin. Demy 8vo. Pp. 118. 1928. Leipzig: Georg Thieme. M. 5.

It is interesting to find the same problems of the surgical responsibilities of the general practitioner exercising the minds of other nations as well as our own. In Germany the man in general practice is not expected to operate, and yet in many

circumstances, especially in the country, he may be obliged to do so—for small things, e.g., the opening of an abscess, or large things, as a strangulated hernia, or in apparently trivial conditions which may lead to fatal consequences, e.g., septic infections of the fingers. This small book is a simply written guide to help such a practitioner to know when he must operate and what he should then do. The matters discussed show practical knowledge and a wise selection both of simple and grave emergency conditions.

Kurze Geschichte der Chirurgie. By W. von BRUNN. Royal 8vo. Pp. 340 +iv, with 317 illustrations. 1928. Berlin: Julius Springer. M. 24; bound, M. 26.40.

THIS is a short but sufficient history of surgery for the use of students, written by Dr. von Brunn, who is professor of the subject in the University of Rostock. The book deals with surgery under five main headings, beginning with prehistoric times, and ending, so far as England is concerned, with Horsley and Macewen. Many of the illustrations are copied from Sudhof and Holländer for the earlier periods and from Garrison for the later ones. They are well reproduced, and any young surgeon who has mastered the work will have laid a sound foundation upon which to build a further knowledge of the work of his predecessors. There is a good bibliography, and two excellent indexes complete the book.

Die Chirurgie: A System of Surgery. Edited by Professors KIRSCHNER and NORDMANN. Fasc. 22, being a part of Vol. II. Imperial 8vo. Pp. 263, with 46 illustrations and 29 coloured plates. Berlin: Urban & Schwartzberg. M. 22.

THIS section is written by Dr. Ascher, formerly at Graz, now at Rottenmann, and is devoted entirely to a consideration of the surgical diseases of the skin and subcutaneous tissues. It is remarkable for the length and detail in which the various subjects are treated, and more especially for the number and beauty of the coloured pictures which illustrate it. After a short historical and anatomical introduction, it begins with an account of congenital malformations, e.g., syndactylism and naevi. In regard to the treatment of the latter, various methods—e.g., excision, carbonic acid snow, and radium—are described, but without a very clear indication as to the relative value of each method. A full account is given of the burns and ulcers resulting from X-ray treatment. Specific infections, both acute and chronic, are next dealt with, and there is a very good account of the rarer diseases, e.g., actinomycosis, mycosis fungoides, and molluscum contagiosum. The next chapter describes the lesions due to faulty circulation and innervation of the skin, e.g., Raynaud's disease, neuropathic ulceration, and certain forms of gangrene. There is a long discussion about the causation and treatment of the chronic congestive ulcer of the leg, and the use of Unna's paste is regarded as the most generally serviceable. Skin-grafting methods are hardly mentioned, but the possibilities of sympathectomy are referred to—rather cautiously.

The concluding portions of this monograph are concerned with the subject of new growths, both innocent and malignant. In addition to a very clear systematic description of each disease, with many beautiful sections and coloured figures, various obscure problems of pathological interest, e.g., metaplastic deposit of bone or calcifying tissue, are discussed. It forms a most valuable article for reference both for surgeons and dermatologists.

Schematische Skizzen zur Einführung in die Chirurgie. By Professor TH. NAEGELI, Bonn. Demy 8vo. Pp. 216, with 300 illustrations, largely in colour. 1928. Leipzig: F. C. W. Vogel. M. 15.

THE leading idea of this most instructive (one is almost tempted to say—amusing) little book is expressed in what purports to be an old Chinese proverb, used as an introductory heading to the preface: "One picture is of more value than many

thousand words." Every aspect of practical surgery is depicted by clear diagrams. For example, the tuberculous abscess, its method of aspiration, the preparation of a slide specimen, the microscopical appearance of the smear, the filling of the cavity with iodoform emulsion—all these fill one page, and descriptive language is almost unnecessary. The book, although small, is tightly packed with instruction conveyed in the most lucid and unforgettable manner.

We confess to a feeling of relief in finding that the strenuous Teutonic mind can permit itself such a pleasant form of relaxation, both in teaching and in learning.

Die Technik des orthopädischen Eingriffs. By Dr. PHILIPP J. ERLACHER, Professor of Orthopædic Surgery in the University of Graz. Royal 8vo. Pp. 482, with 331 illustrations. 1928. Vienna: Julius Springer. Paper covers, M. 44; bound, M. 46.8.

THIS text-book of orthopædic manipulations and operations is very complete, both in the letterpress and in the illustrations. It begins with a short introductory section dealing with the chief diseases which call for orthopædic treatment—e.g., tuberculosis, rickets, and the various forms of paralysis. This is followed by a brief description of the operative principles which are applicable to tendons, bones, joints, and nerves, together with the methods and indications for forcible manipulations.

The bulk of the work consists in a systematic consideration of the various regions of the body in regard to deformities, and the means at our disposal for their correction. In each part of the body a description is first given of the ideal position of rest, and how this may be secured and maintained. Then the various operations and manipulations necessary for the cure of deformity are given, and these are clearly illustrated by very well drawn pictures, the majority of which are original.

Perhaps the most notable feature of the book is the great prominence given to the various operations for the transplantations of tendons and muscles. Transplantation of the pectoralis major to the lower angle of the scapula for serratus paralysis, of the pectoral and latissimus dorsi for deltoid loss, of the abdominal external oblique as a substitute for the gluteus maximus, are examples of this type of operation, which are less known in this country. Payr's method of exposure of the shoulder-joint by turning down the bony origin of the deltoid is also both new and attractive, though one would wish for some critical summary of results obtained by these novel methods.

Another feature of this work which commends itself to us very strongly is the inclusion of many methods which have originated in foreign countries. American, Spanish, Italian, French, and English authorities are freely and faithfully quoted—and the work has thus much more of an international character than most of the modern German works on surgery.

The section on the knee-joint is, we think, rather out of proportion. So much space is devoted to recurrent dislocation of the patella, and so little to the much more important subject of internal derangement of the knee, including the rupture of the crucial ligaments.

The book concludes with a summary of modern literature, a list of authorities quoted, and an index. It is a most valuable addition to surgical literature, being especially useful for purposes of reference.

Diagnostik mit freiem Auge Ektoskopie. By Dr. EDUARD WEISZ, with a Preface by Professor Dr. FRIEDRICH KRAUS. Royal 8vo. Pp. 184 + xi, with 56 illustrations. 1928. Berlin and Vienna: Urban & Schwarzenberg. M. 10.

ACCURATE observation is the basis of medical science. Recent years have seen such a great elaboration of laboratory methods and scientific instruments that there has arisen a danger of medical men relying upon these and neglecting the simple but difficult methods of palpation, inspection, and percussion. It is especially the elaboration of the method of inspection of the patient with the naked eye that is here commended, under the title of ectoscopy, to distinguish it from the many methods of scientific inspection through the prisms and telescopes of endoscopy.

The mechanism of normal respiration is first described, with special reference to the changes in the appearance of the thorax and abdomen. It is claimed that by careful inspection, and by noting the movements of the intercostal spaces, the level of the diaphragm can be seen. Then the phenomena of forced respiration, and especially the sudden inspiration of sniffing or snuffing (*Schnupf-phänomen*), and those of phonation, are described, and it is noted that at the beginning of the act of speech widely separated muscle groups are brought into play quite outside the region of the thorax. Most notable of these is the abdominal musculature. From a surgical point of view, the most interesting observations are those which show the absence of these phonation and respiration phenomena over certain areas of the abdomen after the occurrence of inflammation or perforation.

As a piece of careful observation this little monograph is well worth perusal.

L'Architecture cellulaire normale de l'écorce cérébrale. By Dr. CONSTANTIN VON ECONOMO, Vienna; French edition by Dr. LUDO VAN BOGAERT, University of Brussels. Imperial 8vo. Pp. 177, with 61 illustrations. 1927. Paris: Masson et Cie. Fr. 80.

THIS is a French translation of von Economo's small book on the cerebral cortex. Although it is much smaller and more concise than his large atlas, the average reader will find in it all that he wants. The clearness of the description and the numerous large and excellent illustrations make it very readable, and valuable also as a work of reference. It deals with the structure of the cortex not only from an anatomical but also, as it were, from a physiological point of view. Although very little mention is made of the connections between one part of the cortex and another, one can see that the author has the outlook of a neurologist who is accustomed to study the results of cerebral disease, rather than that of a pure anatomist. The book has been brought fully up to date and can be recommended as the best monograph on its subject.

Radiologie clinique du Tube digestif. By PIERRE DUVAL, J-CH. ROUX, H. BÉCLÈRE. Two volumes. Large 4to. Pp. 340, with 832 illustrations. 1928. Paris: Masson et Cie. Fr. 265.

THIS work appears in two volumes for foreign countries, the first and larger one dealing with the stomach, the second with the duodenum. Each volume treats its subject in a similar manner. First there is a description of the radiological technique, and then the X-ray appearances of the normal organ are described. There is no stereotyped normal shape or position, and the many variations are detailed. This is clearly necessary if the demarcation between normal and abnormal is to be properly understood, and the authors take considerable pains to eliminate such possibilities of error.

The subsequent chapters deal with all the gastroduodenal lesions which can be recognized by X-ray examination. The general plan is to describe concisely the radiological findings in conjunction with numerous plates. These illustrations form a very prominent feature of the book, and are arranged in a manner which is most helpful and instructive. Occupying the whole of the right page are the X-ray photographs, and on the left page there are schematic diagrams of these photographs with short explanatory notes. The result is to make it quite clear, even to those not intimately acquainted with the pathological appearances on an X-ray photograph, where the lesion is, and what is its nature. The authors have taken all these illustrations from living subjects, and the findings have been verified; this large collection has entailed work extending over many years.

We think the title of the book is happily chosen. Radiology alone may be misleading, but when the findings are placed side by side with those elicited by the clinician, it is impossible to exaggerate their helpfulness. It is this intimate co-relationship in the diagnosis of gastroduodenal disease that the authors have sought to emphasize, and in our opinion they have greatly enhanced the value of their book by this eminently practical policy.

The two volumes are attractively got up, and the reproduction of the photographs is excellent. We strongly recommend this work to the notice of clinicians and radiologists alike, and we are of the opinion that it marks a very distinct advance in the application of radiology to clinical medicine and surgery. The authors are men of such distinction that we feel their names alone are sufficient guarantee of the high technical quality, authenticity, and general excellence of the work.

Le Tube Duodénal: ses Applications au Diagnostic et à la Thérapeutique. By MAX EINHORN, Professor of Medicine in the Post-graduate Medical School of New York, etc. Translated from the second edition by GUSTAVE MONOD. Medium 8vo. Pp. 136, with 126 illustrations and 30 plates. 1927. Paris: Masson et Cie. Fr. 25.

THIS seems to be a faithful translation of Einhorn's book on the duodenal tube. Most British surgeons will probably prefer to read the original, but for those who read French fluently, Monod's translation is of a convenient size and cheap. It describes the technique of the use of the tube both for diagnosis and treatment. Readers unacquainted with these methods will be surprised how varied are the uses to which a duodenal tube can be put. There is a final chapter on the intestinal tube, an instrument which has been used to some extent in Germany, but scarcely at all in this country.

Mutationstheorie der Geschwulst-Entstehung. By Dr. Med. K. H. BAUER, A.O., Professor of Surgery in the University of Göttingen. Demy 8vo. Pp. 72, illustrated. 1928. Berlin: Julius Springer. M. 3.90.

THIS is an attempt to give a biological explanation of the origin of tumours. The author reviews the facts of mutation as it is known in plants and animals. His belief is that at some time while active cell division is going on in somatic cells the mutation occurs. By chance this alteration in the gens of one of the chromosomes may confer upon the cell malignant properties which are now transmitted unaltered to future generations of cells. It is a fascinating conception of tumour growth, particularly as worked out by the author of this monograph; but except by hinting that any irritant (since it may start cell proliferation) may predispose to cancer it does nothing to clear up the etiology of this baffling disease. It tends, indeed, to make the reader feel that cancer is almost a natural and inevitable phenomenon and therefore will be always beyond our control.

Contributo alla Chirurgia del Rene e dell' Uretere. By F. DE GIRONCOLI. Demy 8vo. Pp. 130, illustrated. 1927. Bologna: Licinio Cappelli. L. 20.

THIS little book offers a detailed study of a hundred cases from the clinic of Professor Velo, of Venice, and it may be noted that though on the cover the publisher is given as Cappelli, the imprint on the title page is "Venezia: Industrie Grafiche Veneziane". The author does not pretend to make any original contribution to renal surgery, but to demonstrate to the general surgeon how far complete investigation of the renal function, and graphic demonstration of departures from normal renal and ureteral form, by modern methods, may assist clinical examination in diagnosis; how errors may be eliminated, and treatment rendered more exact. It is certainly a true and most creditable record of patient, skilful, and successful work.

Il Pneumorene. By GIUSEPPE NISIO. Medium 8vo. Pp. 135, with 42 illustrations. 1927. Bologna: Licinio Cappelli. L. 20.

THIS monograph with the untranslatable title modelled on 'pneumothorax', and meaning insufflation of the perirenal tissues with gas, is a record of experience from the clinic of the University of Bari. The method, due to Carelli of Buenos Aires, and first described in this country by Hernaman-Johnson, is designed to facilitate

the radiographic demonstration of abnormalities of the kidneys. The pictures substantiate the claims to some extent, but the technique is attended by dangers which, though the author says they are not those of the method but of failure to observe the necessary precautions, will give pause to those considering its adoption. The gas, which should be oxygen (lest it get into the veins), travels along the course of the vessels, and if it be allowed to enter under too great pressure or in too great quantity may be found in the neck or in the popliteal space: it is evidently by no means impossible, even with care, to introduce the gas in quantity into the venous system with dangerous if not fatal results. A bibliography is given, as are some case records and some fairly good illustrative plates.

Surgical Anatomy of the Human Body. By JOHN B. DEEVER, M.D., Sc.D., LL.D., F.A.C.S., Surgeon in Chief, Lankenau Hospital, etc. Imperial 8vo. Second edition, in 3 vols., revised and rearranged. Illustrated. Vol. I., pp. 551; Vol. II., pp. 854; Vol. III., pp. 763. 1927. London: Wm. Heinemann (Med. Books) Ltd. £8 8s. per set.

THIS work should be in the hands of every practising surgeon. It is dedicated to surgeons and to students of surgery and anatomy, and the author in doing so describes himself as a 'fellow-student'. This treatise is evidently not intended for students working for qualifying examinations, but every surgeon knows that when the days of examinations are past his knowledge of anatomy tends gradually but surely to get blurred. Then comes a time when a book such as this is of the greatest possible value in working out details of operations.

The treatise, which consists of three large volumes, is by far the best and fullest ever produced on surgical anatomy: it is amply and beautifully illustrated by drawings from original dissections made specially for this work. Every anatomical point to which a surgeon may possibly wish to refer will be found here, and for the rarer and more complicated operations the assistance given will be invaluable.

The first volume now consists of the surgical anatomy of the head and the brain, the face, mouth, nasopharynx, eye, and ear, so that the surgeon who confines himself to one of the surgical specialities of this region will not have to turn from this volume to find the material he desires. The second volume contains the anatomy of the upper extremities, neck, shoulders, back, and lower extremities; the third volume that of the chest, abdomen, pelvis, and perineum.

The old anatomical terminology is retained in the text, being followed by the English equivalent of the Basle anatomical nomenclature in parenthesis.

Professor Deever is to be greatly congratulated on having produced a book which is such a notable addition to the operating surgeon's armamentarium; in his preface he states that his object has been to produce a work of reference which is, comparatively speaking, complete; and there is no doubt that in attempting this he has been remarkably successful.

Selected Papers on Injuries and Diseases of Bone. By SIR WILLIAM IRELAND DE C. WHEELER, M.D. (Dublin), F.R.C.S.I., F.A.C.S. (Hon.). Demy 8vo. Pp. 148 + xx, with 100 illustrations. 1928. London: Baillière, Tindall & Cox. 10s. 6d. net.

THIS little collection of lectures and essays comes within the class of light surgical literature. It can be picked up and a section read at a time with both pleasure and instruction to the reader, for apart from the value given to it by the experience of its author, there is much to set one thinking. The introduction, ranging widely, is worthy of perusal by every medical man. Note the warning against self-satisfaction quoted from Ambroise Paré: "I have so certainly touched the mark whereat I aimed that antiquity may seem to have nothing wherein it may exceed us, besides the glory of invention, nor posterity anything left but a certain small hope to add some things, as it is easy to add to former inventions." As Sir William says: "He little foresaw Lister and the future in cultivating an attitude so detrimental to progress." We could scarcely find a greater warning against self-satisfaction.

The first lectures are a series upon fractures of the lower extremity, containing much valuable detail, more particularly with reference to fractures of the pelvis and femur. The next lecture contains a description of the 'sleeve' method of amputation, an ingenious procedure for cases of complicated fracture of the femur. Then there are valuable articles upon bone-grafting, including Albee's operation for spinal caries, of which the author is an upholder as a method of treatment in adolescence. Later, Sir William includes "fourteen points about bone-graft", a valuable result of personal experience. The other articles range over subjects of major surgery, such as that of the sacro-iliac joint, down to such a small matter as the operation for hammer-toe, all of them worth reading, and written simply but adequately.

Praktische Orthopädie. By Dr. A. SCHANZ. Royal 8vo. Pp. 560 + x, with 504 illustrations. 1928. Berlin: Julius Springer. M. 42; bound, M. 44.20.

DR. SCHANZ'S name is one that has been known in orthopædic surgery for a very long time. In writing this work he is, as he points out, writing on the experience of thirty years of orthopædic practice, and the book is very largely a dissertation of his own personal methods and views. It is a work which will be studied by the specialist who desires to know these views, rather than by anyone who is studying orthopædies in a more elementary manner. The arrangement is, to us in this country, unusual, and perhaps not very convenient.

In the first section a brief general account is given of the meaning of orthopædic methods in general, and the ends that have been attained. A second rather brief section is devoted to a general account of the diseases treated. Little attention is given to pathology, diagnosis, etc., and the object is rather to say in general what can be done by orthopædic methods for rickets and other bone diseases, rheumatism, including different forms of arthritis, and the various types of paralysis. Only when regional orthopædies is approached in the third section do we find any considerable detail, but here there is a very complete list of deformities, with excellent illustrations by drawings, including many rare conditions, and the inclusion of some of these is the most valuable part of the work. The methods of treatment, however, are almost entirely personal to the author or those approved by him. The description of operative methods tends to be brief and incomplete, and the practical work—operative, mechanical, and instrumental—must be considered to be rather old-fashioned by our standards.

A great deal of space is given up to disease and deformity of the spine, to which Professor Schanz has devoted much time and energy. All orthopædic surgeons know of the enormous amount of ingenuity that has been devoted to the treatment of scoliosis—with, however, it is to be feared, very little practical result. It requires an enthusiast to keep up a continued interest in the mechanical treatment of this very difficult deformity.

Amongst the methods peculiar to the author, the use of bone-pins, inserted to assist in the manipulation and fixation in the correct position of portions of bone during operation and subsequent fixation, is one that deserves mention. Professor Schanz, for example, regulates the angulation of fragments after an osteotomy by this means, and his use of pins for this purpose certainly seems worthy of trial. The book is profusely and very well illustrated.

Clinical Surgery: An Introduction for Junior Students. By J. W. DOWDEN, M.B., F.R.C.S.E., Surgeon to Chalmers' Hospital; Consulting Surgeon to the Edinburgh Royal Infirmary. Crown 8vo. Pp. 68. 1928. Edinburgh: Oliver & Boyd. 2s. net.

THIS is a short essay or sermon on note-taking and early clinical work on the surgical side. In spite of much good advice, it does not take the reader very far—certainly not far enough for the modern student.

Handbuch der speziellen Chirurgie des Ohres und der oberen Luftwege. Edited by Professor Dr. F. BLUMENFELD and Professor Dr. R. HOFFMANN. Fourth edition. Vol. I, Pt. 1. Imperial 8vo. Pp. 600 + vi, illustrated in colour and black-and-white. 1928. Leipzig: J. A. Barth. M. 120; bound, M. 128.

THIS work is the first half of the first volume of the 4th edition of this well-known text-book. It deals with the anatomy only of the head and neck, nose, ear, throat, mouth, larynx, and mediastinum; there is added a section on the lymphatics of these parts.

Much of the volume is taken up with what may be described as the general anatomy of the head and neck, and does not bear directly on the special subjects; it gives the impression of catering for students without a good grounding in anatomy. The same criticism applies to the section on the mouth and on the neck and mediastinum, though much of the latter is made valuable by the use of coloured illustrations of transverse sections through the thorax.

The section dealing with the nose and accessory sinuses, by Professor H. Rhese, is admirable. It occupies nearly one-third of the volume, and is profusely illustrated for the most part by the beautiful and well-known preparations of Onodi, many of them natural size. There are 162 illustrations in the 200 pages. It is perhaps unfortunate that there are not more X-ray pictures—only those of the frontal sinuses are shown. As the result of the arrangement of the volume into sections by different authors there is some reduplication of illustrations.

The ear section is good, and has some very helpful illustrations of the internal ear, notably one of the bony cochlea and one of the membranous labyrinth as it lies in the bone. It is interesting to notice that the pneumatic and diploetic types of mastoid are emphasized, the author (Professor Stenger) stating that the sclerotic type always contains diploetic bone. The variations in the course of the lateral sinus are of interest as bearing on their surgical application. Here again skiagrams of the mastoid and internal ear might well have been included.

The section on the lymphatics is excellent, and is illustrated with photographs of patients showing enlargements of various glands. The sections on the neck, pharynx, larynx, trachea, and mediastinum do not call for special mention.

The book is good, but the range is unnecessarily wide, covering as it does much that cannot rightly be included in the surgery of the ear and upper respiratory tract.

The Pathology, Diagnosis, and Treatment of Neoplasms originating in the Walls of the Urinary Bladder. By L. R. FIFIELD, F.R.C.S. Crown 8vo. Pp. 94 + xi, with 6 coloured plates and 6 other illustrations. 1928. London: H. K. Lewis & Co. Ltd. 7s. 6d. net.

THE proofs of this monograph, based on the essay which gained the Buckstone Browne Prize of the Harveian Society for 1927, had been passed by the author only just before his tragic death in a street accident. It is a careful analysis of a series of 306 consecutive clinical cases of vesical growths admitted to the London Hospital, of which 155 were carcinomas and 151 benign papillomas. Their chief pathological and clinical features are mentioned, and there is an excellent chapter on diagnosis. The treatment of benign growths by 'electro-coagulation', as the author prefers to designate the method, is fully described, and is advocated as the method of choice. Where unsuitable, suprapubic operation is performed, and consists in excision, open diathermy, or partial cystectomy. Transverse incision of the bladder, and transperitoneal cystotomy, are not considered to present any advantage. If papillomatosis involves the whole bladder, total cystectomy is indicated.

For carcinoma partial cystectomy is advised, and if the lower end of a ureter is involved, it is removed along with the growth, and the proximal part transplanted into the bladder through a separate stab wound. Total cystectomy and cysto-prostatectomy, though not performed in any of this series of cases, are occasionally justifiable, and raise the problem of diverting the urine. While the easiest and safest method is that advocated by Watson—bilateral nephrostomy with ligation of the

ureters below the utero-pelvic junction—yet it entails such great discomforts that the ideal method is considered to be the transplantation of the ureters into the large bowel. The various means of effecting this are fully considered in the last chapter, and a comparison instituted between the operations of Stiles and Coffey. An account is given of the author's experimental transplantations of the ureter in 30 dogs and cats, and the conclusion is reached that ascending infection following uretero-intestinal anastomosis may travel either by the lumen or along the lymphatics. Infection by the lymphatics may be prevented in cats and dogs by painting the lower end of the ureter with rectified spirit, while the oblique passage of the ureter through the bowel wall diminishes the liability to infection by way of the lumen.

The monograph bears evidence of very thorough and painstaking investigation.

Fascial Grafting in Principle and Practice. By H. C. ORRIN, O.B.E., F.R.C.S. (Edin.), late Surgeon to Craigleith Hospital, Edinburgh; Ministry of Pensions Orthopaedic Hospital, Newcastle-on-Tyne. Medium 8vo. Pp. 92, with 47 illustrations. 1928. Edinburgh and London: Oliver & Boyd. 7s. 6d. net.

THIS little monograph gives a concise account of the technique and uses of fascial grafting, based upon the practical knowledge of the author. The anatomy of the fascia lata is well described, and the differences in structure at various levels are clearly defined. The closure of the hiatus left after removal of the graft is recommended as a routine. This is eminently sound practice, and often ignored by many operators, probably on account of the difficulty experienced in approximating the edges of a wide gap. Fascial grafting in tendon injuries, contractures, ankylosis of joints, hernia, and so forth is now widely practised. The approval given by the author to the fascial envelopment of an injured nerve trunk will not be subscribed to by the majority of surgeons experienced in this field. Many of the references contained in the short bibliography at the end of the book are incomplete in important details, an omission which tends to mar what is otherwise a carefully written and informative essay.

Handbook of Diseases of the Nose, Throat and Ear. By W. S. SYME, M.D., F.R.F.P. and S.G., F.R.S.E. Second edition. Crown 8vo. Pp. 395 + xvi, illustrated in colour and black-and-white. 1927. Edinburgh: E. & S. Livingstone. 12s. 6d. net.

THE author's purpose has been to give in a concise and practical manner the essentials of the specialty. The fact that the first edition was published in 1920, and that the second is now required to bring it up to date, shows that it has met with some success. In it will be found the views and practice of the author set down somewhat dogmatically, but this, in a students' work, is rather an advantage. In spite of the considerable degree of condensation which has been necessary, the book is eminently readable. It would, perhaps, have been more useful to have devoted some of the space given to the description of operations to differential diagnosis and further clinical detail. The illustrations are many and good, with the exception perhaps of the coloured ones. The subject matter in this edition has been brought thoroughly up to date.

Handbook of Diseases of the Ear. By RICHARD LAKE, F.R.C.S., and E. A. PETERS, M.D. (Cantab.), F.R.C.S. Fifth edition. Demy 8vo. Pp. 310 + xviii, with 80 figures in the text and 4 coloured plates. 1927. London: Baillière, Tindall & Cox. 12s. 6d. net.

THE first edition of Lake's *Handbook on Diseases of the Ear* was published in 1903, and has been recognized for a generation as an extremely valuable little book. The lapse of fifteen years since the last edition has fortunately necessitated considerable addition and alteration. The result, on the whole, has been good, although the difficulty of grafting on the new to the old has not been entirely overcome, giving a

somewhat patchy effect. Thus, methods of treatment such as the use of Lucæ's probe (this being even illustrated) and oto-massage, which have largely fallen into disuse, receive undue prominence. The mind of the reader is not left very clear as to when and when not to operate on nose or pharynx in cases of deafness. The physiology of the vestibular apparatus is well described, but the method of carrying out vestibular tests should have been more precisely laid down. Among additions, the description of aural bacteræmia is worthy of special mention. The illustrations are good, and the coloured plates of abnormal conditions of the tympanic membrane are excellent.

Urography. By WILLIAM F. BRAASCH, B.S., M.D., F.A.C.S., of the Mayo Clinic, in collaboration with BENJAMIN H. HAGER, B.S., M.D., of the Mayo Clinic. Second edition, revised and enlarged. Medium 8vo. Pp. 480, illustrated with 759 roentgenograms. 1927. Philadelphia and London: W. B. Saunders Co. 60s. net.

DR. BRAASCH needs no introduction to the readers of the *BRITISH JOURNAL OF SURGERY*; his various articles on the surgery and pathology of the genito-urinary tract are well known and thoroughly appreciated in this country. His position in the Mayo Clinic, where he is Head of the Urological Section and Professor of Urology, must give him access to an enormous amount of material; we doubt whether there is any urologist who is better entitled to write such a book as that we have now the pleasure of reviewing.

In his preface to this edition he mentions that it is ten years since he published his monograph on pyelography; since that time this method has been extended so as to include the entire urinary tract, and he uses the word 'urography' to represent the various regional terms of pyelography, ureterography, cystography, and urethrography.

The book is published in a handy size, is admirably printed, and the numerous 'urograms' are beautifully produced; the author's style is clear, and printer's errors are only noticeable by their absence. The work contains a vast number of facts, and Dr. Braasch is sometimes content to state the appearances he has observed, and refer the reader to the illustrations for confirmation, without generalizing; this makes the book rather tough reading, and the average reader must, we think, be content to bite off small fragments at a time and digest them at his leisure; it is certainly not a monograph to be read in a hurry.

An attempt to review this work in detail would need many pages, and we shall draw our readers' attention only to a few of the more interesting points; we are convinced that the book will quickly become the standard authority on the subject, and will have to find a place on the overcrowded shelves of the modern surgeon.

In the chapter on 'Technic' we note that Dr. Braasch, whilst convinced of the superiority of the halogens in solving the problem of finding a safe and satisfactory opaque medium, prefers the iodide of sodium to the bromide; he employs it in a solution of 12 per cent. He considers that the syringe method of injection is, in experienced hands, more likely to produce a uniform distention of the pelvis and ureter than the gravity method; he advocates the use of a small ureteral catheter, and thinks that a simultaneous bilateral pyelography is not advisable, though he uses it in exceptional cases. He has found that gas as a medium, while theoretically ideal, has not proved to be of much practical value.

Perhaps the chapters on the diagnosis of stone in the kidney and ureter and that on the diagnosis of renal tumours will prove to be the most interesting and instructive to the average surgeon and urologist; much of this work seems to us to be new and to be unknown to the surgical world.

The article on the normal renal pelvis filled us with admiration and dismay; we admired the completeness of the description of the many variations in form, but we are dismayed because it will be more difficult than ever to say which are normal variations and which are those produced by the invasion of an early renal growth.

We heartily congratulate Dr. Braasch and his collaborator, Dr. Hager, on the production of this very fine monograph, and we do not hesitate to advise our readers

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to study it. We imagine it will be one of those books that will be kept on a handy shelf and frequently consulted by the surgeon who has a difficult 'urogram' to decipher.

Synopsis of Surgery. By ERNEST W. HEY GROVES, M.S., M.D., B.Sc., Surgeon, Bristol General Hospital; Professor of Surgery, University of Bristol. Eighth edition, fully revised and enlarged. Crown 8vo. Pp. 684, with 164 illustrations. 1927. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

GROVES' *Synopsis of Surgery* is so well known that it seems superfluous to do more than mention that a new edition—the eighth—has just been published. The entire text has been revised, and a number of headings brought up to date. The whole has been achieved without adding materially to its size.

A Manual of Surgical Anatomy. By CHARLES R. WHITTAKER, F.R.C.S. (Ed.), Assistant Lecturer on Anatomy, Surgeons' Hall, Edinburgh. Fourth edition. Crown 8vo. Pp. 471 + xii, with 116 illustrations. 1928. Edinburgh: E. & S. Livingstone. 15s. net.

THE first edition of this book appeared in 1910. The subject matter has been well chosen and is excellently arranged; in fact, the amount of information in a compressed but readable form is remarkable. The illustrations aptly supplement the text where necessary. Fig. 3 ("The Sensory Areas of the Head") would be improved by being printed in colours. A few additional diagrams would aid the text, as, for example: lymphatics of the mammary gland; the palatal muscles; the nerves of the anterior abdominal wall; and a composite diagram giving the chief sites for nerve-blocking in the trunk and limbs. The diagram of the chief communications between the portal and systemic veins helps to call attention to points which students often neglect, but it seems to be placed just where it is apropos of nothing in particular. A brief statement of symptoms of obstructed venous circulation and a reference to the Talma-Morrison operation would improve the chapter. Incidentally the hæmorrhoidal arterial terminations (W. E. Miles) might receive mention.

In view of the prominence given to-day to the subject of varicose veins, an illustration of the veins of the lower limb would be justified. The "dimensions of the female pelvis" should be removed from the appendix and put with the account of the genital organs. An illustration of the muscles of the female perineum, to compare with the excellent one of the male, would be an advantage. The radiograms are well chosen and reproduced. One or two figures showing the internal architecture of the long bones would be an asset to the appendix on the anatomy of the long bones. The tables of muscles and their nerve supply are useful.

There are a few misprints and questionable spellings, but these are minor blemishes which will doubtless disappear in future editions in a book of obvious merit.

The Glasgow Medical Journal Centenary Number, 1828-1928. Edited by JOHN PATRICK and GEO. A. ALLAN. Medium 8vo. Pp. 73-224, with 21 illustrations. 1928. Glasgow: Alex. Macdougall; London: H. K. Lewis & Co. 5s.

THE *Glasgow Medical Journal* reached its centenary in February. The editors, to celebrate the event, have issued a well written and well illustrated double number which is divisible into two nearly equal parts. The first deals more strictly with the history of the journal and with medical journalism of the last hundred years. The second part treats of medical practice in Glasgow since the journal was founded. The information is conveyed in a series of articles contributed by writers who have an intimate knowledge of the subjects which have been allotted to them. Where all is good, perhaps the most useful from the historical side is that by Dr. John Henderson on "The Position of Extramural Teaching in Glasgow in 1828 and its Progress during the Century." Extramural schools have never taken root in England, so that it has been somewhat difficult for an Englishman to understand the position they held and still hold in Edinburgh and Glasgow. Dr. Henderson's

article clears up the difficulties, and he points out both their advantages and disadvantages. Dr. Oliphant in another article deals with the Royal Faculty of Physicians and Surgeons of Glasgow. He states that until 1850 every new member joining the faculty was obliged to subscribe to a Widows' Fund, entailing an expense of at least £150. This amount was prohibitive to most young practitioners, and it was only those who were comparatively well off who could afford to become members. Further, the mortality from typhus and other fevers was very high, so that many died without leaving any dependents who might benefit from the fund.

The last article is by Dr. Fergus, who gives pleasantly and anecdotally his recollections of medical Glasgow and its University.

This centenary number is adorned with twenty-one illustrations which are well reproduced; many of them are portraits of those who have rendered good service to the journal in their generations, but have now fallen upon sleep.

Chemotherapeutic Researches on Cancer: with especial Reference to the Lead and Sulphur Groups. By A. T. TODD, M.B. (Edin.), M.R.C.P. (Lond.), Hon. Assistant Physician, Bristol Royal Infirmary. Medium 8vo. Pp. 127. 1928. Bristol: J. W. Arrowsmith Ltd. 2s. 6d. net.

SELENIUM has long had a reputation for effecting a diminution in the rate of cancerous growths. Its action, however, has been capricious, though it certainly has some analgesic effect. It occurred to Dr. A. T. Todd that a combination of lead and selenium might be of service. With the help of a strong committee of chemists and doctors he has been carrying out researches on a lead-selenide compound, the complete results of which are embodied in this book.

The Bristol Royal Infirmary allocated 10 beds for the purpose of treating advanced cancer cases. The expense of the researches was generously defrayed by Sir Gilbert Wills and Mr. G. F. Hutchings, and all the strictly chemical work was done under the supervision of the Professor of Chemistry of the Bristol University and his assistant.

The pharmacology of the drug has been thoroughly worked out—its toxicity, its abortifacient action on rabbits, its distribution in the tissues, and its affinity for cancerous growths; the tables for the latter, however, are somewhat disappointing.

The action of the drug has been tested on both animal growths and man. Mice and rat sarcomata were used; and the effect in some cases was striking. Among the human cases are some which show a regression of the tumour, and many in which the drug caused a relief from pain. In others it had little effect, and in those cases with secondary growths in the liver it appeared quite useless.

Complete details are given of the manufacture of the stable compound lead-selenide, and protocols of all the animal experiments are set out in full. The research also ranges over similar drugs with a tellurium and sulphur basis.

It is the best review so far of the biochemical aspect of malignant growths.

The Art of Anæsthesia. By PALUEL J. FLAGG, M.D. Fourth edition, revised. Medium 8vo. Pp. 384 + xviii, with 135 illustrations. 1928. London: J. B. Lippincott Co. 21s. net.

As the title indicates, this work on anæsthesia is essentially a practical one, and the present edition well maintains this feature. The author has not found it necessary to make any changes in the first eight chapters, but the ninth, which deals with nitrous oxide and oxygen, has been brought up to date as regards general considerations and details of technique. The section dealing with spinal and local anæsthesia has been amplified, but cannot be considered to have received quite as much attention as this important branch deserves; on the other hand, the difficult question of choice of anæsthetic has been reconsidered and very fully and carefully discussed in the revised last chapter. We feel that this book deserves a wide circulation amongst practical anæsthetists.

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Le Drainage en Chirurgie Abdominale. By F. M. CADENAT, Paris, and M. PATEL, Lyon. Medium 8vo. Pp. 412. 1928. Paris: Gaston Doin et Cie. Fr. 18.

THE authors state that there are now two schools of thought among abdominal surgeons. On the one hand, some surgeons hold that drainage is always desirable; and on the other, many believe that drainage can be dispensed with, once a source of infection has been eliminated. The authors believe that the truth lies between these two extremes, and in support of their view they produce a carefully balanced reasoned argument.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXVII (Vol. VII, Fourth Series), No. 2, April, 1927. Royal 8vo. Pp. 127-252, illustrated. 1927. London: Lancet Ltd. 12s. 6d. net.

THE last quarterly number of these Reports contains two articles at least which are of interest to the general surgeon. One is "The Pathology of Achalasia of the Cardia", in which the view is advanced that the disease is due to inflammation and destruction of Auerbach's plexus. This opinion is based on histological examination of several cases. Mr. Slesinger contributes an article on "Peri-arterial Sympathectomy", in which he gives the indications for, and technique of, the operations, and some of his results.

The quarterly number for January, 1928, contains nothing of surgical importance.

Surgical Anatomy. By GRANT MASSIE, M.B., M.S.(Lond.), F.R.C.S., Senior Demonstrator of Anatomy and Surgical Tutor, Guy's Hospital Medical School. Pp. 413, with 121 illustrations, some in colour. Medium 8vo. Pp. 413 + viii, illustrated. 1928. London: J. & A. Churchill. 15s. net.

THE author, the artist, and the publishers may be congratulated very heartily on having achieved the chief object in view—to present the facts of surgical anatomy correlated with their clinical application in such a way that a student can learn them in a pleasant, educational manner, without hurry or cram.

The general setting and arrangement of the work leaves nothing to be desired. The text is well expressed, and the descriptions are clear. Here and there perhaps a little more clinical matter could be introduced with advantage—for example, by illustrating the areas of hyperæsthesia in abdominal lesions; short notes on local analgesia and nerve-blocking would also help to focus the student's attention on the distribution of the sensory nerves. These are very well depicted by thick lines in the diagrams relating to this part of the subject. The account of the surgical anatomy of the wrist and hand is particularly good. Bankart's posterior approach in cervical rib might be mentioned. A transverse section through the neck below the level of that in *Fig. 24* would help. A little more might be added on the clinical signs of flat-foot. Illustrations of the lymphatics of the mammary gland and axillary region, and one or two showing the catchment areas of the inguinal glands, would improve the book. *Fig. 97* may illustrate, but does not explain, the inguinal operation for femoral hernia. It would do so if combined with a window view of the structures or with a modified *Fig. 96*.

Misprints are very few. The index is excellent. The facts shown in *Figs. 119* and *120* should be briefly indexed. There is no list of illustrations, which is unusual.

Taken as a whole, we have nothing but praise for this book. It would be difficult for any surgeon to read it without learning something fresh, and it should certainly be bought by those who do any teaching or demonstrating.

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EPOCH-MAKING BOOKS IN BRITISH SURGERY.

BY SIR D'ARCY POWER, K.B.E., LONDON.

VII. SEVERALL CHIRURGICALL TREATISES BY RICHARD WISEMAN, 1676.

THE treatises of Mr. Serjeant Surgeon Wiseman mark an epoch in the history of British surgery. They stand midway between Elizabethan surgery and the dawn of modern surgery as represented by the writings of Cheselden and Percivall Pott. Wiseman looked back to the younger Clowes and to John Woodall just as Pott mentions with respect the treatises of Wiseman himself. Like Woodall and all his predecessors, who became consulting surgeons, Wiseman gained his experience in land and sea warfare. He seems to have served in the Dutch Navy, afterwards in the service of King Charles I at the battle of Worcester, at Dunbar, and in the West of England. When the Commonwealth made England uncomfortable for Royalists he served as a surgeon in the Spanish Navy. He was the last of the great English surgeons who graduated through this rough school, and he was the first consulting surgeon in the modern sense of the term to obtain a large civil practice in the more settled times after the Restoration. Wiseman rarely saw a patient unless he came with a recommendation from another medical man, and he was always careful to associate himself with a physician to advise him in the treatment.

Wiseman's surgical treatises differ from the earlier books on surgery because they are written for the enlightenment of the more educated surgeons who had come into existence during the last twenty or thirty years of his life. They make no attempt to cover the whole ground of surgery, but deal with special subjects in the light of his own experience. Each section is pre-faced with a short account of the disease under consideration, a definition, the cause so far as it was known, the signs, the prognosis, and the treatment. Details are then given of the individual patients whom he had treated—more than six hundred in number—and of the results obtained, and it is noteworthy that he records his failures as well as his successes. The histories are extremely

interesting. They illustrate contemporary practice and the extreme difficulty of getting the patients to submit to the knife; cautery and escharotics being the alternatives. They show that the surgeon was completely subservient to the physician, and draw attention to the inroads on surgical practice made by the unlicensed practitioner, the bonesetter, and the lady bountiful. All is written in good English and forms instructive and often amusing reading.

Wiseman gives the following history of a case in which he extirpated both tonsils:—

“A person of honour, aged ten years, having been some years diseased with praeternatural swellings in her tonsils, with great defluxion upon her lungs, also difficulty of breathing, and at last of swallowing, her drink frequently running out at her nose, was brought to London and put into the hands of Sir Fra[ncis] Prujean and Dr. Luke Rugely. Mr. Arris was surgeon to the family. Various remedies had been prescribed; but they proving ineffectual to remove them I was consulted and proposed the extirpation of them by actual cautery. But that not being admitted I undertook it by escharotics and came the next day prepared and placed her in a clear light, her head leaning to a bedpost, Mr. Arris sitting behind, holding it steady. I began with the left tonsil; holding her tongue down with a speculum *ingulae* while I pressed into the body of the tumour with a long-pointed Caustic-stone fixed to a stick. After I had held it awhile I pulled it out and with a fir-stick dipt in *oleo vitrioli* pressed into the same place, then pulled out that too and gave her a glass of water to wash her mouth. I repeated the use of the several caustics; she washing her mouth always after the use of them. By this method I penetrated into both the Tonsils and divided the left into two. In the time of working with the caustic I dried up the salts of them with armed probes so well that her mouth was not fretted in any part by them and after I had finisht the work I washed the tonsils with decoct. *hordei* and she afterwards gargled with a pleasant julep of distilled water, *acet. ros.* and *syrup. diamoron*. Having divided the one tonsil and diminished the other I caused an instrument to be made to convey a ligature about the basis of them, with design to tie and cut them off one after the other. I began with the left tonsil passing a ligature upon the one half and cut it off close. There happening no disturbance by bleeding I tied the other and cut it off; then with a *Vitriol-stone* I rubbed the remaining basis and thereby smoothed and cicatrised it in 4 or 5 days. I then began with the right tonsil, Mr. Arris holding her head as formerly. I made a ligature upon the basis of it and holding the end of it with my right hand I passed a pair of probe-scissors with the other hand close by the said basis and supposing I had encompassed the root I clipt; but it not coming away I thrust my scissors downwards and cut again, being confident I had clipt through the fleshy body. I pulled the ligature but it not coming I was somewhat surprised what should be the cause; therefore, pulling my seissors a little back and turning them more off from the basis, I clipt again. I cut off the bigger part of it and brought it out with my ligature; at which instant she choked. The Ladies seeing it shrieked, and became troublesome. I desired Mr. Arris to sit still while the patient choked, she gaping, I passed into her throat the instrument I had made the ligature by, and raising up the



RICHARD WISEMAN

remaining excrescence she recovered and cried, Shall I live? I replied Yes, if you sit still. I passed another instrument fitted with a ligature. I, changing the instruments, the excrescence slipped down and she choked again; but I raised it up and relieved her; then tied the ligature upon it and looked into her throat; where I saw it rooted like a Cock's comb and that in endeavouring to cut the tonsil close by the root I had cut below the part I had tied. Which if I could have foreseen and have passed my scissors close a little lower, one snip had brought it clear away, but not supposing it rooted so low and working blindfold, I was at a loss. But being now better informed I passed my crooked Probe-scissors down lower, cut it clean off by the roots and brought it away with my ligature. I cicatrised it with the Vitriol and Alum-stones in a few days. Yet somehow after she was cured a part of her drink came out at her nose; which happened by reason of the tonsils drawing that part of her palate down lower than naturally it should; and I suppose that was the cause of her speaking so. But some while after she was freed of those inconveniencies." It is clear from this that Wiseman was not very far from the discovery of adenoids.

There was "A Gentleman, some years since, very curious and neat in wearing his hair who was persuaded to permit it to be boiled as it grew on his head; by which doing an erysipelas was raised in the hairy scalp. I let him bleed in the arm and purged him with lenients; all things else were done by revulsion; but, by reason of his hair, there were no applications made to relieve the part affected; whereupon a suppuration followed; which put me upon a necessity of cutting it off and applying fomentations discutient and drying. I met with much difficulty in curing the ulcer, but at last it was cured as a corrosive ulcer."

The difficulties of travelling were still so great that the surgeon was often required to remain with his patient until he was either cured or out of danger. Wiseman alludes to it incidentally in telling of the following case: "A child about two months old laboured of some indisposition of body and was observed to have a swelling in the right cod. In pressing upon it with my fingers it seemed to me not to lie loose in the scrotum; it yielded to a moderate pressure; and being somewhat reduced, the residue of it jerked suddenly up with a noise, which confirmed it a hernia intestinalis by relaxation. I took the compass of his loins to the groin and the next day brought him an emplastron ad herniam with some bolster-trusses. I reduced the rupture as easily as I had done the day before, then, applied the Emplaster, fitted the bolster [pad] over it and tied it moderately straight [tight]. These trusses had the straps stitched on behind; they were brought between his thighs and fastened to the upper part of the bolster cross each other by points. I showed the nurse how they were to be put and gave her caution lest the bolster should at any time gall the thigh or scrotum; or that by too strict a bandage the hip should be hurt or by too slack a bandage the hernia should slide down under the bolster. Having thus forewarned her I made my visits the seldomer, as not desiring to appear too officious, and indeed was fetch the next day to a patient some miles off where I stayed three weeks. At my return I made a visit to this little one and felt the rupture slipt down into the scrotum by reason of the slackness of the bracer; and the nurse had pulled the straps between the

thighs so over-streight as to bring the bracer down and would not believe it could be kept up without a bandage from the shoulders. But I reduced the rupture, tied it streighter, slackened the straps between the thighs and showed her it could not fall over the buttocks whilst it was so tied. The next error she committed was in bracing it too streight about the hips, whereby she had galled it on the right hip, and then she complained to her Lady that it was not possible to cure the child without giving it some healing drinks; such and such had been cured so in a few days. To this I replied that the cure consisted in keeping up the rupture; that done it would cure without drinks; that there was but little to be given to such an infant. Yet I wished them to consult their physician therein. I applied an Emplastron diachaleiteos upon the excoriation with two or three folds of soft linen over it and braced the bandage slacker; by which dressings the excoriation cured, the Nurse became more experienced and the child was happily recovered in a few weeks, but left not off the wearing of the truss till the summer following."

Wiseman's honesty and independence are well illustrated in the following case: "A Lady coming to town with a swelling in her left breast consulted some of our profession and at last me. She said she had some years since kernels in her breast which were judged the King's Evil, upon consideration of which she was presented to His Majesty and touched. In progress of time they swelled and her breast being extremely painful, she desired my judgement of it. The swelling was large and round and greatly inflamed under which it was soft and seemed to have matter in it. The parts more distant were hard and several tubercles lying under the skin made it unequal; yet the breast was not fixed. She urged me instantly to deliver my thoughts of it; which to decline, I turned from her and told her friend it was a cancer, and that I saw no hopes to save her life but by cutting it off. He wished me to consider how I delivered such judgement of it, two Chirurgeons having lately assured her to the contrary, they taking it for a phlegmon [inflammation]. But I, not being used to guide my judgement by what others delivered, confirmed to him what I had before said by a sad prediction, which befel her a few weeks after. And, indeed, there was no way then to deal with it but by cutting off her breast."

He speaks tolerantly of quacks and bonesetters. He says of quacks: "A person of quality aged about fifty years of a plethoric body had a wart upon the first joint of one of her forefingers. It was imprudently undertaken by some pretender to surgery and treated as ill; so that after many months endeavours he was dismissed and a more knowing Chirurgeon entertained who found much difficulty in the cure." In another case: "A man about fifty years old of a gross body frequently afflicted with the gout and labouring then under it in his left leg (as I remember) was commended to an Empirick who pretended extraordinary skill in the cure of that disease but understood no more than what he had learned by books."

Of bonesetters he says: "In several observations in this book I have had occasion to take notice of the inconveniencie many people have fallen into through the wickedness of those who pretend to the reducing luxated joints by the peculiar name of Bone Setters; who, (that they may not want employment) do usually represent every bone dislocated they are called to

look upon ; though possibly it be but a ganglion or other crude tumour or preternatural protuberance of some part of a joint. In which cases their rash extensions do frequently cause sad accidents. But their more gainful way is by extending and dressing up joints rather wrenched than dislocated ; in which, if they escape undetected, they must needs reap great credit, the patient recovering so soon the ease and use of them. Whereas if the joints happen to be really luxated scarce one of them knoweth how to reduce them."

Wiseman had a large and fashionable practice during the most dissolute period of Court life after the Restoration. His treatise on Gonorrhœa, therefore, is of especial interest as throwing light on the contemporary methods of treating venereal disease—and truly men and women paid in full measure for their pleasure. Little distinction was made between the different forms of infection ; the nature of a urethral stricture was not understood ; bougies were employed, but the catheter was not yet in general use. The patients put off treatment as long as possible and only called in the surgeon when they had retention or extravasation, and they were then bled, purged, vomited, and salivated as a matter of routine.

The portrait of Wiseman here reproduced hangs in the Council Room of the Royal College of Surgeons of England. It was painted by Sir Balthazar Gerbier (1601 ?–1667) a friend of Rubens and a protégé of George Villiers, Duke of Buckingham. A man of great versatility, he was amongst the first to suggest in 1641 the establishment of 'mounts' or banks combining pawnbroking with banking business.

THE RESULTS OF A NEW OPERATION FOR THE SUBSTITUTION OF A THUMB.

By J. LEONARD JOYCE,

HONORARY SURGEON TO THE ROYAL BERKSHIRE HOSPITAL, READING.

IN 1917 I designed a new operation to substitute a ring finger of the opposite hand for a thumb lost as a result of injury or disease, and afterwards described a case in which the operation had been done successfully. Ten years have elapsed since the finger was transplanted, and another operation of the same kind has been performed. This paper records the present state of the first and brief details of the second case, and concludes with some remarks on the various methods of reconstructing the mutilated thumb, and a bibliography.

[PARTICULARS OF THE TWO CASES.

Case 1.—The original case was fully reported in the *BRITISH JOURNAL OF SURGERY* (1918, v, 499). The excellent result then recorded was marred after some months by a flexor contracture at the interphalangeal and metacarpophalangeal joints. So persistent



FIG. 293.—Case 1.



FIG. 294.—Case 1.

was this contracture and so tiresome to control that, no voluntary movements being possible at the interphalangeal and the metacarpo-phalangeal joint of the new thumb (distal and proximal interphalangeal joint of the old finger), I determined to ankylose these joints in a position of election—namely, a



FIG. 295.—Case 1.

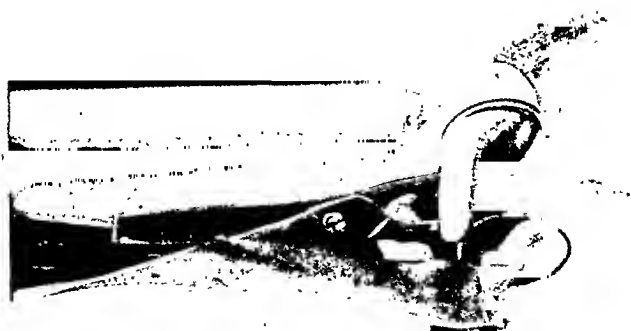


FIG. 296.—Case 1.

slightly flexed position at the metacarpo-phalangeal joint and a slightly hyperextended position at the interphalangeal joint. To do this an operation was performed on Feb. 1, 1921. Both joints were exposed through short longitudinal incisions over their dorsal aspect, and small wedges of bone and articular cartilage were removed from the adjacent bones. The wounds

healed by first intention, and soon afterwards the bones united in their chosen positions. From this time the new thumb has been an unqualified success. For seven years the patient has possessed a thumb ankylosed in hyperextension at the interphalangeal joint and slight flexion at the metacarpophalangeal joint, but having a complete range of normal movement at the carpo-metacarpal joint.

PRESENT CONDITION.—The circulation of the thumb is excellent, its nail grows at a normal rate, and there is no evidence of any trophic change. Pin-prick and light touches of cotton-wool are felt everywhere and are localized correctly. By trade the man is now an upholsterer: he uses his thumb naturally and says it feels to him as if it were his old thumb. To the various small injuries common to an artisan's life, such as slight burns, blows, and cuts, the new thumb has reacted in a normal manner, and there is a hard callosity on the ulnar side of the proximal phalanx caused by the friction of his cutting-out scissors. I have related how the thumb behaved to the trauma of a surgical operation. *Figs. 293–296* show the condition of the thumb in June, 1923, while engaged in various active occupations.

Case 2.—The second case is that of a man, age 32, who received a shell wound of his right hand in March, 1918, resulting in the total loss of its radial side, including the thumb and index finger with their metacarpal and carpal bones. When he came under the writer's care in April, 1919, there was fibrous ankylosis of the wrist-joint in palmar flexion, and considerable ulnar deviation of the remnant of his hand. Active and passive movements of

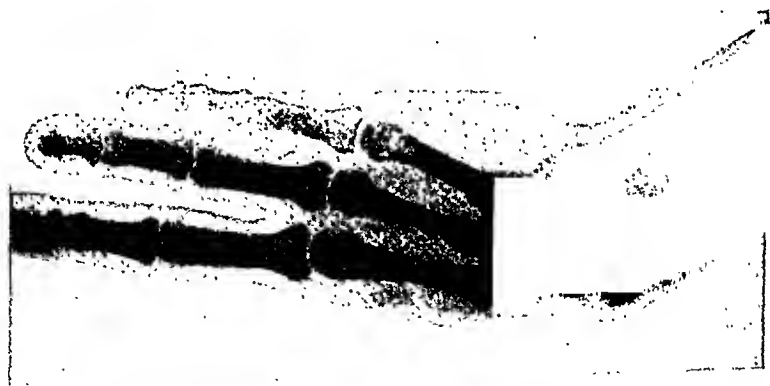


FIG. 297.—Case 2. Radiogram of hand in April, 1919, before reconstruction, showing loss of radial side of hand and ulnar deviation.

the three remaining fingers—middle, ring, and little fingers—were normal. The styloid process of the radius projected on the radial side of his wrist, and was covered by a thin and adherent scar. The radiogram showed an arthritis of the wrist-joint and loss of the proximal row of carpal bones.

The problem was how to restore some function to a hand mutilated and left useless by the loss of its radial half but retaining three useful fingers on its ulnar side. The idea was conceived that if a new thumb could be articulated to the projecting styloid process of the radius, some useful function

might be obtained. Dorsiflexion of the wrist, correction of the ulnar deviation, and replacement of the adherent scar over the radial side of the hand by healthy skin, were all necessary before the idea of providing a new thumb could be entertained.

The scar on the radial aspect of the wrist was excised widely, and replaced by healthy skin and subcutaneous tissue from the abdomen by means of a pedicle graft. The graft had healed soundly by the end of May, 1919. Dorsiflexion of the wrist and correction of the ulnar deviation were then obtained easily by means of a succession of plaster casts.

By February, 1920, pin-pricks were felt everywhere in the grafted skin, and I decided on trying to provide a new thumb by substituting the ring finger of the opposite hand. The thenar muscles of the right hand were lost entirely: to obtain the essential movements of the thumb seemed impossible. The intention was to provide a fixed sensitive thumb to which he could oppose one at least of his remaining fingers, and in this way procure some use in his hand. Measurement proved that, because of the shortened hand caused by the loss of the proximal row of carpal bones, the proposed new thumb, if articulated to the styloid process of the radius, would have almost normal length proportionately to the remaining fingers.



FIG. 298.—Case 2. The two hands growing together. The ulnar deviation and palmar flexion of the right hand have been corrected. April, 1920.



FIG. 299.—Case 2. Radiogram of hand after reconstruction. June, 1920.

Misgivings were felt about the chance of success because of the deep scar, and the necessity of making an incision through grafted tissues. The risk of failure was explained to the patient, and he was told that, were the operation successful, the new thumb would be fixed. Encouraged by the success of my first case, he decided to have the attempt made.

The operation described by the writer in the *BRITISH JOURNAL OF SURGERY* was performed, but the new thumb was fixed to the exposed tip of the styloid process of the radius instead of to the trapezium; and as there were no thenar muscles, the bed for the reception of the new

thumb was prepared in the fatty tissues grafted from the abdomen. The first stage of the operation was performed on Feb. 20, and the second stage on April 19, 1920. There were no complications.

PRESENT CONDITION.—The new thumb shows little evidence of trophic change. The skin is slightly glazed and smooth and is apt to become blue



FIG. 300.—Case 2. June, 1928.

in cold weather, but pin-pricks are felt everywhere and localized correctly. Cotton-wool touches are perceived with proximal localization. Sometimes the sensation of itching is experienced, and the patient volunteers the state-



FIG. 301.—Case 2. June, 1928

ment that, if not thinking, he will scratch to the radial side of his hand in the position his old thumb once occupied. The joint between the styloid process of his radius and the metacarpal bone of his new thumb (proximal phalanx of his ring finger) is perfectly stable, and allows about half the extent of

passive movements possible in the carpo-metacarpal joint of a normal thumb. The metacarpo-phalangeal joint of the new thumb (proximal interphalangeal joint of the old finger) has become contracted towards the flexor aspect to a right angle, and only a few degrees of passive flexion and extension can be obtained. The interphalangeal joint of the new thumb (distal interphalangeal joint of the old finger) is not contracted, and allows a few degrees of passive movement. The terminal phalanx and the nail are deformed and smaller than normal, the result of a burn contracted a few months after the operation. The sore caused by the burn healed normally and has remained healed for seven years.

The aim of the operation—the provision of a fixed sensitive thumb to which he could oppose the remaining fingers of his hand—has not been realized; partly owing to the flexor contracture at the metacarpo-phalangeal joint, and partly owing to an initial error in technique, whereby the finger was transplanted with its transverse plane nearly parallel to that of the palm of his hand instead of at right angles to it. I have no doubt that by a second operation at the present time both faults could be rectified, and the original intention of the operation accomplished. The patient is, however, a busy man and unwilling to give up the time necessary to complete the reconstruction. The result is relatively successful considering the difficulties of the case. *Figs. 297–301* show the original condition of his hand, one of the various steps in its reconstruction, and its present condition.

COMMENTARY.

Much attention has been devoted to the reconstruction of the mutilated thumb. The methods employed may be classified as follows:—

1. *Methods entailing the transplantation of tissues from a distance*:—
 - a. Pedicle graft of skin, tubed, and stiffened by a free bone-graft.
 - b. Transplantation of a toe.
 - c. Transplantation of the ring finger from the opposite hand.
2. *Methods using the remaining parts of the injured hand*:—
 - a. Making a cleft between 1st and 2nd, or between 2nd and 3rd, metacarpal bones—called by the ugly name ‘phalangization’.
 - b. Finger transplantation.
 - c. Rotation of two of the remaining fingers.
 - d. A combination of two or more of the above methods.

It is not my intention to discuss these various procedures in detail. When confronted with a case of mutilated thumb a surgeon will use his judgement as to which method best fits the particular case in question. A few remarks, however, may have some value.

As far as the author can gather from the papers which have been written on the subject, his are the only cases of loss of a thumb and the whole of its metacarpal bone which have been treated by a method falling into the first group. The transplantation of tissues from a distance has been chiefly employed when at least a part of the first metacarpal bone has been present.

Of the second group, the combined method used by Perthes seems to have given a good result when employed for the total loss of a thumb including

its metacarpal bone, but certainly no better than that obtained in the first case recorded in this paper. In the method of Perthes the index finger of the injured hand becomes the new thumb; when this finger is already stiff through injury or disease, especially if it hampers the movements of the other three fingers—which has been the case in some of the patients on whom this type of operation has been practised—its conversion into a prehensile digit seems to be the obvious and right course to pursue. On the other hand, when the index finger of the injured hand is perfectly normal, its sacrifice for the sake of a new thumb would be a greater one than that of the ring finger of the opposite hand. From the point of view of function of the donor hand, as long as the metacarpal bone is not interfered with and except in special professions, the loss of the ring finger is of little moment.

Better function is the prime object for which these operations should be undertaken. In the author's second case it is possible that a better functional result might have ensued by adopting one or a combination of the operations from the second group, but this is uncertain, as the patient has been unwilling to have the reconstruction completed.

Since operating on the cases recorded in this paper the writer has seen a third patient who had lost by an accident his right thumb and its metacarpal bone. There was a fine flexible scar on the radial aspect of the hand, the thenar muscles were present and could be actively contracted and relaxed, and the rest of his hand was normal in every respect. A good functioning thumb at the expense of the ring finger of the opposite hand could have been assured. Unfortunately he was unwilling to endure having his hands joined together for several weeks.

Whether there is any value in the suggestion made in my original paper that "by suitable modifications in the details of the operation any less part of a thumb can be similarly replaced by substituting for it a part of a finger of the opposite hand" I have had no chance of putting to the test, but the excellent functional result in the original case and the way the thumbs have stood the test of time in both cases lead me to believe that, when practised for the condition for which it was designed, the operation of substituting for a thumb the ring finger of the opposite hand gives a valuable functional result.

SUMMARY.

1. A new operation for the substitution of a thumb was described in the *BRITISH JOURNAL OF SURGERY* in 1918 (Vol. v, p. 499), with details of a case in which the operation had been performed successfully. The history of the case has been brought up to date.
2. A second case presenting great difficulties is recorded in which the operation met with a modified success.
3. The new thumbs have stood the test of time.

BIBLIOGRAPHY.

- ARANA, G. B., "Phalangization of the First Metacarpal", *Surg. Gynecol. and Obst.*, 1925, June, 859.
 ARLT, B. R., "Daumenplastik", *Wien. klin. Woch.*, 1917, xxx, 15.

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- DUNLOP, J., "The Use of the Index Finger for the Thumb: some interesting Points in Hand Surgery", *Jour. of Bone and Joint Surg.*, 1923, Jan., 99.
- HUECK, H., "Einfall von Daumenersatz durch einen unbrauchbaren Finger", *Deut. Zeits. f. Chir.*, 1920, cliii, 321.
- HUGUIER, *Arch. gén. de Méd.*, 1874, i, 78.
- JOYCE, J. L., "A New Operation for the Substitution of a Thumb", *Brit. Jour. Surg.*, 1918, v, 499.
- LAMBERT, O., "Résultat éloigné d'une Transplantation du gros Orteil en Remplacement du Pouce", *Bull. et Mém. Soc. de Chir.*, 1920, xlv, 689.
- LENORMANT, C., "Le Traitement des Mutilations des Doigts et en particulier du Pouce par les Autoplasties et Transplantations", *Presse méd.*, 1920, 223.
- LYLE, H. H. M., "The Formation of a New Thumb by Klapp's Method", *Ann. of Surg.*, 1914, lix, 767.
- "Deformity of the Hand—Formation of a New Thumb from Stump of First Metacarpus", *Ibid.*, 1921, lxxiv, 120.
- "The Formation of a Thumb from First Metacarpus", *Ibid.*, 1922, lxxvi, 121.
- "Treatment of Disabilities of the Hand", *Ibid.*, 1923, lxxviii, 816.
- MACHOL, "Beitrag zur Damenplastik", *Beitr. z. klin. Chir.*, 1919, cxiv, 181.
- MANASSE, P., "Vorstellung eines Falles mit Daumenersatz und Fingerauswechsellung", *Berl. klin. Woch.*, 1919, lvi, 717.
- MUELLER, W., "Anatomische Studien zur Frage des Daumenersatzes", *Beitr. z. klin. Chir.*, 1920, cxx, 595.
- NICOLADONI, C., "Daumenplastik und organischer Ersatz der Fingerspitze (Anticheiroplastik und Dactyloplastik)", *Arch. f. klin. Chir.*, 1900, lxi, 606.
- "Weitere Erfahrungen über Daumenplastik", *Arch. f. klin. Chir.*, 1900, lxxv, 695.
- NUZZI, O., "Intermetacarpolisi distali chirurgica", *Riforma méd.*, 1921, xxxvii, 248.
- OMBREDANNE, M. L., "Constitution autoplastique d'une Pouce prenant au moyen du 1er Metacarpien", *Bull. et Mém. Soc. de Chir. de Paris*, 1920, xlv, 158.
- PERTHES, V., "Ueber plastischen Daumenersatz insbesondere bei Verlust des ganzen Daumenstrahles", *Arch. f. Orthopädi. u. Unfallchir.*, 1921, xix, 198.
- PIERCE, G. W., "Reconstruction of the Thumb after Total Loss", *Surg. Gynecol. and Obst.*, 1924, xxxix, 259.
- SCHPELMANN, E., "Das spätere Schicksal einer Daumenplastik", *Zeit. f. orthop. Chir.*, 1919, xxxix, 181.
- SCHMIEDT, W., "Beitrag zur Daumenplastik", *Deut. Zeit. f. Chir.*, 1916, cxlv, 420.
- SPENCER, W. G., "Plastic Operations on the Thumb", *Med. Sci. Abst. and Rev.*, 1920, iii, 29.
- VERRALL, P. J., "Three Cases of Reconstruction of the Thumb", *Brit. Med. Jour.*, 1919, ii, 775.

CRETINISM.

BY R. LAWFORD KNAGGS,

CONSULTING SURGEON TO THE LEEDS GENERAL INFIRMARY.

CRETINISM is a condition characterized by dwarf-growth associated with defective mental development. It is met with amongst the offspring of goitrous or cretinous parents in certain districts where goitre and cretinism are endemic; also, sporadically, in goitre-free districts, when its subject is often the child of healthy parents whose family history shows no taint of goitre or of cretinism. Both forms depend upon an insufficient supply of the secretion of the thyroid gland, are benefited by thyroid treatment—sometimes in sporadic cases almost to the point of cure—and are closely allied to the myxœdema of adults, in which the thyroid deficiency declares itself *after* the skeleton and the brain have been developed normally.*

The study of cretinism involves the consideration of three definite and distinct conditions:—

1. *Endemic cretinism.*
2. *Sporadic cretinism.*
3. *Cachexia strumipriva.*

An absence or inadequate supply of thyroid secretion is now generally accepted as the cause of the symptoms in sporadic cretinism and cachexia strumipriva, but there is some difference of opinion as to the exact position which deficient secretion occupies in the etiology of endemic cretinism. There is no question as to the intimate resemblance between the endemic and sporadic varieties, and in both the disease develops in infancy or early childhood; but the onset of cachexia strumipriva, which follows complete removal of the thyroid and is also closely akin, usually occurs in adult life, and only a proportion of cases, viz., those in which thyroidectomy has been carried out during the growing period, come within the scope of this article.

It is often stated or assumed that the thyroid gland is absent in sporadic cretinism; but palpation, which is the only method of investigation that can be employed during life, is not reliable, and even failure to find the gland at an autopsy is not infallible. Serial sections of the cervical tissues have shown that traces of ectopic thyroid tissue may be found when the gland itself is missing. This fact, and the not infrequent existence of accessory thyroids, make it difficult to be sure that, in any particular case, there is absolutely no

* The 14th 'conclusion' in the Report of the Committee of the Clinical Society of London on Myxœdema runs as follows:—

"(14) That a general review of the symptoms and pathology leads to a belief that the disease described under the name 'myxœdema' as observed in adults is practically the same disease as that named 'sporadic cretinism' when affecting children; that myxœdema is probably identical with cachexia strumipriva; and that a very close affinity exists between myxœdema and endemic cretinism."

thyroid tissue capable of functioning. Though total absence of the gland has been demonstrated in a few cases by the serial-section method (aplasia of the thyroid; thyreo-aplasia), in many sporadic cases some kind of a thyroid gland is certainly present, though it shows evident signs of mal-development or of atrophic degeneration (thyreo-hypoplasia).*

There are other cases, too, usually of later origin, in which the signs of cretinism supervene upon some infectious illness, such as whooping-cough, measles, etc. The atrophic change in the gland in these cases has been compared to atrophy of the testes after mumps, but it is more probable that the margin of the resistance of the gland to degenerative changes has been whittled away by the lowered vitality resulting from the recent debilitating sickness.†

Occasionally it may not be easy to decide whether a cretin is of the endemic or the sporadic kind. An infant may be born without a thyroid in a neighbourhood where goitre and cretinism are endemic. The thyreo-aplasia would point to sporadic cretinism, but the district, and perhaps also the ancestry, might be in favour of the endemic disease.

But a cretin, whether endemic or sporadic, is hardly ever a cretin at birth. Usually the first recognizable signs of the disease make their appearance within the first or second year, sometimes even later. In such cases, when the infant's thyroid is unequal to its needs, this period of grace is attributed to the action of the maternal thyroid secretion conveyed to the foetus during intra-uterine life, and very possibly the mother's milk may act as the medium after the child is born.

It is not known to what extent the foetus has to depend upon its mother's thyroid secretion under normal circumstances, but there is ground for belief that the foetal thyroid does not begin to function till a comparatively late period of intra-uterine life.¹ If, therefore, the mother's thyroid gland should chance to be the subject of cretinoid degeneration—not necessarily of a goitre—the interval previous to the onset of symptoms will probably be shortened. It is under such circumstances, when the thyroid secretion fails in both mother

* Packard and Hand's case was a clear sporadic cretin 6 years of age. Post mortem the thyroid was fibrotic; the alveoli were small; many contained no colloid, and those that did showed it in very small amount. (*Amer. Jour. Med. Sci.*, cxxii, p. 293.)

Aschoff's case of a 6-months-old child had a cystic tumour at the root of the tongue, and a vesicle of the size of half a pea at the site of the lateral lobe, which he regarded as a remnant of the bronchial pouch. (Falta, *The Ductless Glandular Diseases*, Meyer's trans.)

In Fletcher Beach's case, age 2½ years, there was no thyroid. (*Trans. Pathol. Soc.*, 1873-4, xxv, 265.)

† Dr. Fagge's second case, a girl age 16½ years, until she was 8 years old "was a good-looking child with a large quantity of black hair. She was lively and good tempered and played like other children" and had made satisfactory progress at school. "When 6 years old she had a slight attack of measles, but did not keep her bed; about the same time she had whooping-cough. Two years afterwards she had a second attack of measles. She then kept her bed for two weeks; she was not insensible. She is further stated to have had erysipelas at that time. Sores followed on the head, and she lost her hair in patches. She suffered from severe diarrhoea, and also from shortness of breath."

This illness seems to have been 'beyond doubt' the starting point of the development of the cretinous state, for she did not grow from that time, and her features changed completely. (*Med. Chir. Trans.*, 1871, liv, 159.)

Magnus-Levy observed one case in a group of endemic cretins who developed fairly normally up to the 10th year, and showed the first signs of cretinic degeneration after an attack of pertussis. About the 16th year rapid degeneration with myxœdema set in. (Falta, *Ibid.*, 167.)

and foetus, that a *congenital cretin* results, i.e., the foetus is born cretinous (*Fig. 302*); such cases are very rare. But a child born of a healthy mother, even if it has a cretinoid father, can still fall back upon its mother's secretion, and the cretinous state will probably not develop till some time after birth, when, owing to degenerative changes, the secretion from its own thyroid is failing. The cretin in that case would be *hereditary*—but not *congenital*.²

'Infantile myxœdema' is a term often met with in the literature of cretinism. It leaves with the reader a sense of uncertainty as to whether it is used merely as a synonym for cretinism or not. The following authoritative statement by Falta³ therefore will help to make its employment clear: "Light cases, or those in which delay in development sets in at a later stage, are to be counted as infantile myxœdema. In severe cases that occur quite early the assumption of a thyreo-aplasia is better warranted." (Elsewhere this author speaks of infantile œdema making its first appearance in the fifth or sixth year of life.)

It is perhaps unnecessary to point out that athyreosis—a word that is frequently used, especially in foreign literature—includes not only many cases of sporadic cretinism, but also those of eachexia strumipriva (operative athyreosis).

The symptoms of cretinism are the consequence of a suppression or retardation of the ordinary course of development. The essential cause of the check is failure of the supply of thyroid secretion. The various organs and tissues feel the loss of its influence in a greater or less degree, and their functions suffer accordingly. The skeleton reacts to the absence or withdrawal of this influence in a remarkable manner, and the resulting changes in endochondral ossification lead to one of cretinism's most striking features—dwarfism.

FIG. 302.—Kocher's 1-year-old congenital cretin. (See text.) (After Kocher, *Deut. Zeits. f. Chir.*, 1892, "Zur Verhütung des Cretinismus und cretinoid Zustände".)

The changes in the bones, and their influence upon the growth of the skeleton, will be considered in their clinical and pathological aspects, and from the important view-point of treatment. But first, some explanation must be given of a circumstance in the earlier history of cretinism which for a long time introduced an element of confusion into its pathology and diagnosis. Virchow, after an investigation of the changes in the skull of a foetus which belonged to the category of 'so-called foetal rickets', came to the conclusion that the case was one of cretinism and described it under that designation. Virchow's 'newly-born cretin' has acquired a remarkable notoriety

in the literature of the subject, for in later years it was shown to be an example of chondrodystrophia foetalis or achondroplasia—one of the diseases which has emerged from the composite group of 'foetal rickets' (Weygand and Bayon—see Wegelin⁴). The point on which Virchow went astray was synostosis of the basis cranii (i.e., of the os tribasillare), which he regarded as 'the central point of the whole disturbance', and, consequently, the great feature of the cretin skull. But it is now known that this synostosis is a characteristic feature of achondroplasia, and that in cretinism the exact opposite prevails, namely, long delay or failure in the obliteration of the spheno-occipital synchondrosis. Virchow's great reputation endowed his error with a long life, and even yet it cannot be said to be quite dead.* To it may be attributed the impression, which at one time was prevalent, that foetal cretinism was not very uncommon, but usually resulted in a still-birth. But it is the foetus affected with a severe degree of achondroplasia that is born dead, whilst the child destined to become a cretin is, except in very rare instances, to all appearances normal for the first few months of its life.

THE CLINICAL ASPECT.

Though differing in non-essential points, endemic and sporadic cretinism are so much alike that one description is usually made to do duty for both. The impression conveyed, therefore, appears to lose some of its lucidity when the reader finds the two conditions very definitely separated in their further consideration, and he is sometimes at a loss to know whether reference is being made to one or both. At the risk therefore of some redundancy a description of each will be given.

The Endemic Variety (*Figs. 303, 304*).—This account is taken chiefly from Osler and Macrae's *System of Medicine* (vi, 449).

The unfortunate sufferers are frequently dwarfs, their height, when full grown, usually varying from 40 to 60 in., and sometimes being even less. The body is short and broad, the neck short and thick, the abdomen large and pendulous. The forehead is low and broad, often sloping backwards, and the skin covering it is thrown into wrinkles. The nose is flat, the nostrils are conspicuous, and the eyes small and widely separated. The expression is stolid. The muddy skin, suggesting the Eskimo, looks œdematous but does not pit on pressure. The legs are short and sometimes crooked, and their muscles weak and ill-developed, so that the gait is feeble and waddling, and may be limited to creeping. The supraclavicular fossæ contain cushions as in myxœdema. The genitals remain infantile, but may develop between the thirtieth and fiftieth years, though rarely to a stage to make procreation possible. In severe cases thick blubbery lips encircling an enlarged and protruding tongue may produce a semi-bestial aspect which is repulsive in the extreme.

"Idiocy is an essential part of cretinism."

"Individuals with less complete bodily and mental change are spoken of as semi-cretins"; and there is a slighter grade still, known as cretinoid,

* In the last edition of a popular text-book of medicine the basi-occipital and basi-sphenoid are stated to be prematurely ossified in cretinism.

in which, with the faec and bodily conformation of the cretin, there is some degree of enfeeblement of the intelligence, and speech is somewhat impaired.

"Cretins are always apathetic and stupid, and lacking in memory and decision. They are not easily excited, but are sometimes vindictive". Some cases can be trained to simple duties, and become hewers of



FIG. 203.—Endemic cretin; high grade. (After Iphofen.)

wood and drawers of water. "These, occasionally spoken of as 'beast men', have the mental development of a trained house dog (Koehler); others, less developed, are spoken of as 'plant men'."

Sporadic Cretinism (Figs. 305, 305A).—

This form, said to be the only one now met with in England, was described by Hilton Fagge⁵ in a well-known communication to the Royal Medico-Chirurgical Society:—

"The body is stunted, the height scarcely exceeding 4, 3, or even 2 feet in different cases. The head is round; the face is broad; the eyes are widely separated by the flat root of the nose; the alæ nasi are thick; the nostrils are rounded; the mouth is very large and generally wide open; the lips are thick; the hands and feet, as well as the fingers and toes, are short and broad. When sporadic cretinism is congenital, it is also attended with a deficiency in the mental powers, varying in degree, but of a character very like that which belongs to the endemic form of the disease. The child is free from the mischievous tendencies displayed by so many idiots. It is good humoured and torpid—often sitting for a long time quiet in one place. Sometimes it can walk only with the assistance of a chair. It is not rarely deaf and dumb. Sporadic cretinism, instead of being associated (like the endemic cretin) with goitre, appears to be attended with a wasting or absence of the thyroid body."

Fagge's first case was the 8-year-old son of healthy parents who lived at Rotherhithe. Their other children were healthy. He was large at his birth, began to cut his teeth at 2 years of age, and first attempted to walk at 3½. Nothing was noticed amiss with the child in early infancy, but afterwards he would sit down when he



FIG. 304.—A cretin (endemic) from Wiesenbrum, age 18 years. In order to estimate the relative length of the arms it may be mentioned that the subject is represented sitting upon a chamber utensil on which he was placed because he was continually dirtying himself. There is an indication of this in the picture. (After Virchow, *Gesammelte Abhandlungen*, Fig. 35, p. 953.)

could, and remain silent for hours in one position. He also ceased to grow, and was thought not to have grown since he was $2\frac{1}{2}$ years old. At the time of the report, though 8 years old, he was more like a child of 2 or 3, very quiet, rarely moving of his own accord, and wearing an air of torpid contentment—though said to have rare explosions of temper. He smiled when noticed, appeared not to know many words, but named things shown to him correctly, and was clean in his habits. His height was 2 ft. $7\frac{3}{4}$ in. and his weight 25 lb.



FIG. 305.—A sporadic cretin, A. C., age 18 years and 8 months, at beginning of thyroid treatment, Dec. 25, 1892. Height $33\frac{1}{2}$ in. (Dr. John Thomson's case, from *Edinburgh Medical Journal*, Feb., 1894.)



FIG. 305A.—Same case as Fig. 305, after 12 months' thyroid treatment, Dec. 20, 1893. Height $37\frac{1}{2}$ in.

There is a great sameness in the clinical histories of these cases, as will be seen from that of the adult which furnished Dolega⁶ with material for his pathological observations.

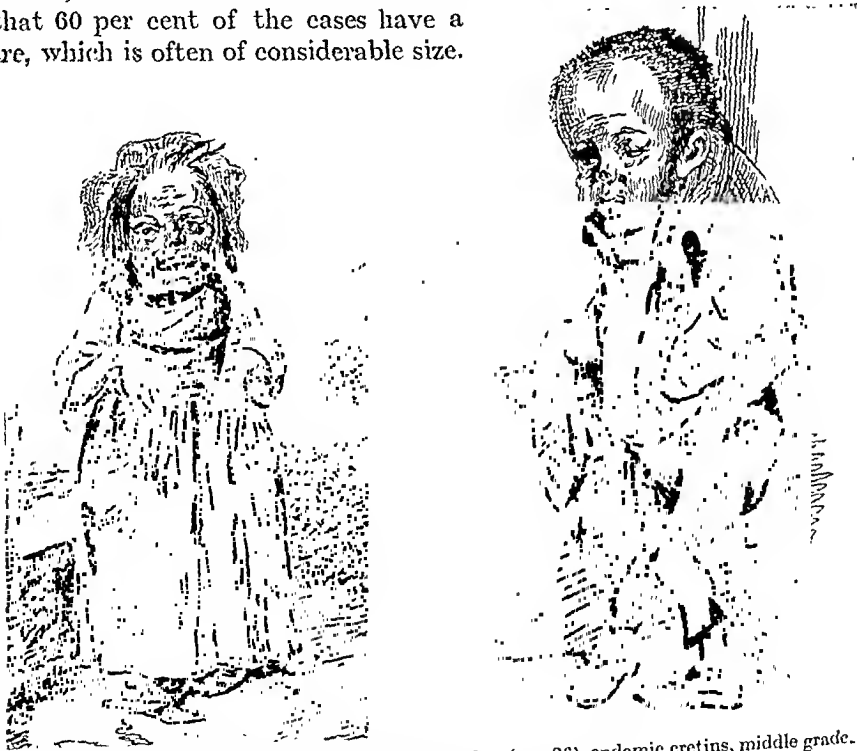
Julius W., born in 1859, of healthy parents and having two healthy sisters, died at the age of 28. There was no similar dwarf in the family or in the village. He was a very small child but thrived normally till he was 9 months old, when his development became very backward. He learnt to walk in his 8th year and by the 10th he could cross a room upright and without help. But the ability to walk was soon lost, and he passed his life in a chair or lying down at intervals. His speech came late, and he could only say short words, such as Papa, Mama, Yes, No. He had fair use of his arms: he passed his excretions at times beneath him, but he was accustomed to show his need by a definite noise. He was fed on fluids because

he did not masticate solids, and up to his last year would play alone with a certain sort of weariness. Admitted to hospital for eczema of the scalp he died suddenly ten days later following diarrhœa and dyspnœa.

These extracts not only give a good general impression of the cretinous individual, but bring out the tendency to an increased severity of the disease in the sporadic variety, as shown by its effect on the stature, the intellectual faculties, and the muscular strength. The difference will be even more evident when the duration of life is considered, but mention must first be made of several other conditions.

The presence of symmetrical fatty masses above the clavicles, external to the sternomastoids, was pointed out by Curling⁷ in 1850, and emphasized by Fagge in his communication. These swellings are now recognized as a distinctive feature in cretinism, especially in the sporadic form, and are a local evidence of the tendency to fat deposition which is present in cretinism and myxœdema. They may disappear in the later (wasting) stages preceding death (Fletcher Beach's case⁸). They are sometimes spoken of as if they were simply myxœdematous swellings, but they are really collections of fat. In both Curling's cases the fat was unencapsuled; in one it was superficial, and in the other it passed down behind the clavicles and filled the axillæ.

Goitre is common in endemic cretins (*Figs. 306, 306A*). Falta⁹ says that they are almost always goitre carriers; Fodere,¹⁰ that the greater part of the children who will become cretins are born with a small goitre the size of a walnut, and Hector Mackenzie¹¹ tells us that 60 per cent of the cases have a goitre, which is often of considerable size.



FIGS. 306 and 306A.—A sister (age 21) and brother (age 26), endemic cretins, middle grade. Note the goitres. (After Dr. Hermann Demme.)

On the other hand, in sporadic cretinism an enlarged thyroid is a rarity. The gland is either absent or in a state of atrophy, though Fagge mentions one case—a boy of 14—in the York County Hospital, who had a large bronchocele, was of stunted growth and deficient in intelligence. His parents had resided all their lives in York, and none of his relations was known to have been an idiot or to have suffered from goitre.

Umbilical hernia is of frequent occurrence in both forms of the disease. It is probably connected with the protuberant distended abdomen and the constipation, which are such pronounced symptoms. The interesting point is that under thyroid treatment not only do the two latter disappear, but the hernia in many cases also gets well.

A diminution in the temperature, and the rate of the pulse and respiration. may be regarded as part of the torpor which is brought out so well in Fagge's descriptions. They may in rare cases become a very striking phenomenon. In a sporadic case recorded by C. W. Townsend, of Boston,¹² "the low temperature, slow pulse and respiration all pointed to the sluggish, almost hibernating condition, which at times was so exaggerated that the child appeared to the mother as if dead". Rarely the slowing of the respiration may be remarkable. Falta mentions a case of Magnus Levy's, aged 28, in which the thyroid had been removed fourteen years before, who breathed only six times in a minute.

Myxœdema.—Though the term 'infantile myxœdema' is supposed to be applied to cases in which the onset of the cretinous state is late, yet a swollen œdematous condition of the skin and subcutaneous tissue, which does not pit on pressure, is undoubtedly present in sporadic cases at an early age. Sidney Phillips,¹³ reviewing a number of the earlier recorded cases, pointed out that it was present in the majority and was to be looked upon as a usual symptom. In the endemic disease, however, myxœdema is not always present, at any rate, in the earlier stages or slighter degrees. In 60 per cent of cases examined by E. Bircher it was absent (Falta¹⁴), and Phillips refers to the statement of Fodere, quoted by the Sardinian Commission, that "cretinous infants mostly become œdematous". Also the latter author writes, "those who have no goitre have characters from which one recognizes they will be in that class" (i.e., endemic cretins). "They are swollen, voluminous, especially about the head and hands".¹⁰ The fact is further emphasized by the case of two cretins observed by H. Bircher¹⁵ in which tetany and severe myxœdema occurred after the removal of their goitres.

The formation of the mucin with which the tissues are infiltrated is due to a change (mucinoid degeneration) in the ground-substance of the connective tissue, which at a certain stage of myxœdema is increased in quantity. The myxœdematous process has been studied in connection with the skin more particularly in the adult disease. It is thought to be inflammatory because of the cell infiltration in and around the sebaceous and sudoriparous glands and in the neighbourhood of the hair follicles.¹⁶ Eventually the myxœdema tends to disappear, the connective tissue becoming infiltrated with white fibres and fat. The supraclavicular swellings are an expression of this stage, which sometimes may be succeeded by marasmus, when the localized swellings may disappear.

The duration of life in most cases of endemic cretinism is somewhat shortened, as might be expected where the intelligence is so defective, but the condition is not incompatible with long life. Kocher has reported cases of 70 and even 100 years, and others still older are on record. Stoccada's cases, which furnished material for his histological illustrations, some of which, through his courtesy, are reproduced here, were 27, 30, 35, 40, and 57 years old respectively. But in sporadic cretinism the danger to life is definitely greater. Bourneville and Bricon's cases were 28, 24 (Obs. vi*), and 31 years old respectively; and Dolega's, the base of whose skull is shown in Fig. 307A, was 28.

According to Horsley¹⁷ the disease beginning in early childhood "reaches its height by the end of 14 or 15 years, so that by the 20th or 21st year it has attained complete development and henceforward remains perfectly stationary until death. Hence at the age of 30 the physical appearance is that of a young child, and the intellectual condition similarly does not advance beyond that of childhood." Further on he writes as if severe cases do not survive beyond 30 or 40 years, but that milder cases may.

Sporadic cretins show a great resemblance to one another, even though they may come from different countries. Endemic cretins, on the other hand, differ from one another much more. Even when they belong to the same family the likeness is not so marked as that between sporadic cases which are not related. There is often more than one cretin in a family—sometimes there may be several. This would seem more likely to occur in the endemic form than in the sporadic; but even in the latter it is not very rare. Fagge refers to three cretins, members of a family of twelve belonging to healthy parents, and Herman¹⁸ reports two instances in which there were three cretins in a family. Other examples may be found in the literature of the subject.

Cachexia Strumipriva.—Only a limited group of cases of this condition can be really compared with endemic and sporadic cretinism, namely, those in which the whole thyroid was removed during the period when the bones were growing, and before the intellectual faculties were adequately developed. Such cases cease to grow, and their epiphyses, or some of them, may remain cartilaginous for years, or for life. Their mental development also stands still. After the growth of the skeleton is completed, total thyroidectomy is followed by a condition which is "probably identical with myxœdema."

THE MACROSCOPIC APPEARANCES OF THE BONES.

Professor C. Wegelin,¹⁹ of Bern, is convinced that "the persistence of the cartilaginous synchondroses beyond the growth age, and the inadequate development of the primitive cartilage-absorbing medullary spaces", are the distinctive marks of the abnormal ossification of cretinism. These features

* According to Falta the oldest sporadic cretin recorded was Bourneville's (Pacha de Bicêtre), age 36. Stoccada quotes the case as 37. There seems to have been some error here, for in Bourneville and Bricon's article (*Arch. de Neurol.*, 1886, xii, 293) Pacha de Bicêtre's case is given in Obs. vi. He was born Aug. 20, 1861, and died Nov. 18, 1885, so that he lived 24 years.

are characteristic of both the endemic and sporadic forms, and also of cachexia strumipriva; and any difference that exists is one of degree only—the degree being least in the endemic disease. The peculiarities of the bones and of their growth, as seen in actual cases and specimens of the three conditions, are described below. It is the endochondral ossification that provides the most convincing evidence of deterioration, but there are signs that membranous bone formation also suffers in some degree.

THE SKULL.*

The well-marked cretin skull presents points of considerable interest. In severe degrees of the disease, especially in the sporadic variety, the departures from the normal are striking. It retains in adult age the peculiarities at the base which characterize the skull of a child at birth. The skull of Dolega's 28-years-old case (*Figs. 307, 307A*) may be cited as an example. It was not



FIG. 307.—The skull of Dolega's case: a sporadic cretin, age 28. (After Dolega, *Zeigler's Beitr. z. pathol. Anat.*, 1891, ix, 489.)

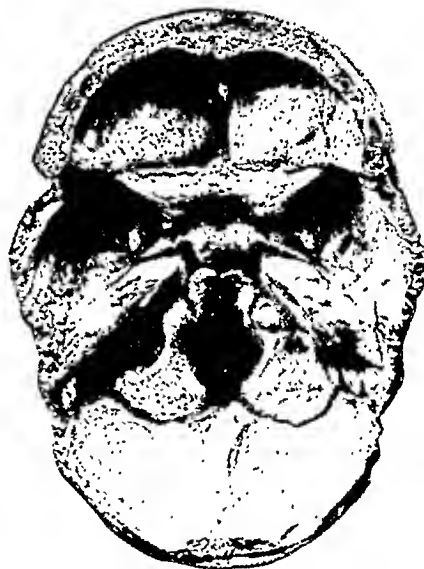


FIG. 307A.—The base of the skull of Dolega's case.

only infantile in its proportions, but its infantile character was further shown by the persistence of all the sutures and synchondroses, by the wide-open anterior fontanelle, and by the presence of those sutures and fissures which suggest the formation of the tabular part of the occipital bone from four centres.

The most notable abnormality was in the base behind the anterior fossa. This part was flat. The occipital squama behind the inferior curved line was reared upwards, but the parts of the occipital in front of that line lay nearly horizontal, and there was no kyphotic curve of the base, as the basi-occipital

* I am indebted for much of the material in this section to a paper by Dolega,²⁰ who made a very careful study of the skeleton of a sporadic cretin aged 28. The clinical history of the case is given on p. 375.

made a very obtuse angle with the body of the sphenoid. The synchondrosis between the pre- and the post-sphenoid centres of ossification had disappeared, but the spheno-occipital (basilar) synchondrosis was completely preserved, and a continuous cartilaginous covering extended from it along the whole dorsum sellæ to the cartilaginous posterior clinoid processes (Virchow's overlying cartilage of the clivus) (Fig. 307A).

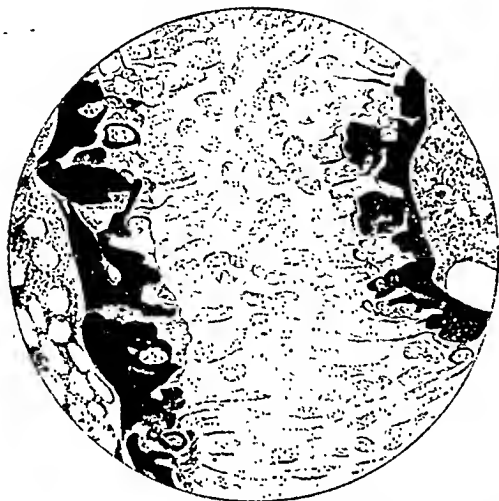


FIG. 308.—Showing a section through the upper part of the spheno-occipital synchondrosis from a 30-year-old endemic cretin (Bosshard). Moderately marked proliferation of cartilage cells in the vicinity of the bone; cell rows present here and there. Few modullary spaces in the bony boundary lamella, which is thinner on the occipital than on the sphenoid side. (After Stoccarda.)

was normal, but the jugular and nerve foramina were contracted.

Though relatively very big for the body, the skull was very short for a person of 28, and this depended on the limitation of the growth of the occipital bone, the basi-occipital portion being strikingly small and much shorter than normal.

A few less important points may be mentioned. The anterior fossa was diminished by the upward arching of the orbital plates of the frontal bone, which left between them a deep furrow in which the cribriform plate of the ethmoid lay. The crista galli was largely cartilaginous, and the sharp points and edges at the base were rounded and swollen. There was also considerable thickening of the vault seen in horizontal section (1.5 to 1.2 cm.), and the mastoid process was absent.

The occipital bone was composed of four parts, like that of a new-born child—the basi-occipital, the two exoccipitals (all unusually small), and the tabular portion. These parts were quite distinct and separated from one another by intervening cartilage. On the under surfaces of the exoccipitals there were no articular processes, but simply flat surfaces for articulation with the atlas. The antero-posterior diameter of the foramen magnum

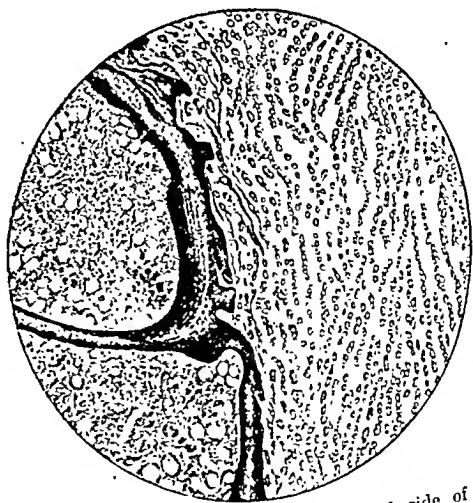


FIG. 309.—Showing the occipital side of the spheno-occipital synchondrosis of a 44-year-old cretin (Christener). Lower part; very slight proliferation of cartilage cells close to the bone; cell rows absent. The bone forms a continuous layer along the cartilage from 0.1 to 0.3 mm. thick. There are no modullary spaces in the bony layer. (After Stoccarda.)

The face looked mean, chiefly owing to the shortening of its upper part. The root of the nose lay further back than usual, and the frontal bone inclined considerably forwards above it. The upper jaw protruded, the arch of the palate was absent, and the alveolar process was broad and high. The two halves of the lower jaw were completely separate and the angles slightly marked.

Nearly all the *milk teeth* present were carious, and the permanent ones seemed to be struggling for the necessary room to develop.

In this country museum specimens illustrating cretinism are rare. In St. Bartholomew's Hospital Museum is the skull of a female sporadic cretin, aged 12 years (No. 286b), who died of bronchopneumonia ten days after thyroid treatment had been started. It is very similar to that just described. The following account is taken from the catalogue.

"The brain case is well developed. . . . Those bones which arise in membrane are as a whole much more completely developed than those arising in cartilage . . . yet even here there is evidence of locally arrested development." (Anterior fontanelle wide open, lateral fontanelles not quite closed.) "The cartilage bones on the contrary are imperfectly developed: the bony units of the basis cranii remain to a large extent independent; there is no shortening of the basis cranii and no synostosis In the occipital the ill-developed basi-occipital and condylar masses resemble those of a very young child, while the upper part of the bone is thick and firm. The root of the nose is broad and flattened, but not drawn in. Dentition is backward. None of the milk teeth have been shed, though the permanent teeth can be seen in the jaws, some of them in abnormal situations." The patient had shown "no abnormality at birth but signs of defective development became apparent after six months. She had never walked or talked, and in her 12th year was 33 inches in height, and weighed 32 lbs. Her external appearance was characteristic of cretinism, the body was hairy, and fatty tumours were present above the clavicles." After death the thyroid gland was found to be replaced by a little fatty tissue.

But this pronounced infantile form of skull is not found in every cretin. In the less severe grades the cranium approximates to the normal, and only the basilar synchondrosis may persist. I am indebted to Dr. D. M. Greig for details of three skulls in the Museum of the Royal College of Surgeons of Edinburgh from sporadic cretins of 21, 13, and 19 years of age respectively. They show no particular abnormality, but the basilar suture is present in all three, as it might have been in healthy individuals of similar ages. In the last of the three—a feeble, undersized, and undeveloped girl—the basi-occipital is flat and horizontal and the occipital condyles are unusually flat. The jugular fossæ are small, the anterior arch of the atlas is not united, and the posterior is deficient for several millimetres.

Though the basilar synchondrosis normally persists long after the other synchondroses have closed, there is no doubt that its ossification is very markedly retarded in both forms of cretinism. It was specially selected by Stoccarda for examination on this account, and it was present in the six cases, ranging from 27 to 57 years of age, upon which his paper is based. (*Figs* 308, 309.)

THE LONG BONES.

It is characteristic of cretinism that the ossific centres are very late in making their appearance in the cartilaginous epiphyses (*Figs* 310, 311), and

when they do they grow slowly.* Growth may come practically to a standstill in sporadic cases, and the epiphyses may remain largely cartilaginous to the end of life. But in the endemic disease, though growth is slow, it is not as a rule interfered with to such an extreme degree, so that, though the epiphysial discs persist some years longer than usual, it is common in adults to find the epiphyses united to the shafts, and only traces of cartilage left in some of the epiphysial regions. Length growth comes to an end when the epiphysial cartilages disappear, and their persistence in these cases explains why, when cretins are submitted to thyroid treatment, growth may take place, particularly in sporadic cases, long after the ordinary growth age has been passed. Some of these facts are illustrated by the following cases.

In the 28-years-old sporadic cretin (Dolega's), to which reference has already been made, there were no ossific centres in any of the epiphyses of the three long bones of the upper extremity, though search was made for some of them with a microscope. In the femur centres were present in the head and the great and small trochanters, and except for a tolerably large ossifying nucleus the lower epiphysis (condyles and epicondyles) was wholly cartilaginous. There were no ossifying centres, also, in the epiphyses of the tibia and fibula, and in the scaphoid and internal cuneiform amongst the tarsal bones.

In Stoeckada's first case (G. M.), an endemic cretin of almost the same age (27 years), there remained traces of the upper epiphysial disc of the humerus, but none of the lower one, and none of the radial and ulnar epiphyses. At the lower end of the femur the marginal part of the disc persisted, but in the tibia and fibula and in the phalanges and metatarsal bone of the great toe no cartilage could be recognized.

The long bones of a cretin are, of course, much shorter than those of a normal-sized person, and they are also shorter in proportion to the length of his own body or his spine. Consequently his limbs are similarly shortened; but the latter disparity might easily be overlooked. It does not resemble in the least the great disproportion between the limbs and the trunk seen in achondroplasia. Moreover, these bones have two peculiarities which are interesting because they are an indication of the muscular feebleness usually associated with the cretinous state: (1) the ridges and projections for

* NOTE ON THE DELAY IN THE APPEARANCE OF THE BONE NUCLEI IN CRETINISM.—Wegelin's inquiry into this subject led him to the opinion that delay in the appearance of the bone nuclei is more striking than the restriction of length growth. He found that in endemic cretinism the appearance of the bone nuclei is considerably delayed, and the delay lasts several years, but is not regularly seen in all bone nuclei. Those of the upper extremity are more delayed than those of the lower (Bircher), and the delay is less in cretinoids than in full cretins. In the neighbourhood of Berne there is a notable absence of bone nuclei, which he looked upon as the expression of a very prevalent failure of ossification, connected probably with thyroid influence. He was confirmed in this opinion by some observations he made upon school children. His investigation was concerned with the lower femoral epiphysis, because the date of the appearance of its nucleus is more reliable than that in the other epiphyses, and in medical jurisprudence its presence is considered a fairly trustworthy sign that the foetus is mature. It was found that there was a marked difference in point of time in the appearance of the bone nuclei in children from goitrous and goitre-free regions. Children from the goitrous districts who were apparently normal (not cretinous) showed a noticeable delay in 60 to 90 per cent of all cases. (*Cor.-Blatt. f. Schweiz. Aerzte*, 1916, xlv, 616.)

muscular attachments are very poorly developed, and (2) though atypical curves are present in some bones, the normal anterior curve of the femur is absent, and the bone is quite straight, suggesting that the lower extremities have not been much used in walking or in otherwise supporting the trunk.



FIG. 310.—Hands of a cretin, age 5 years, showing the absence of ossific nuclei.
(Dr. R. A. Veale's case. Radiographs by Mr. C. Guy Whorlow.)



FIG. 311.—Hand of a normal child, age 5 years, to compare with Fig. 310.

THE SPINE.

The evidence of delayed endochondral ossification is well marked in the spine. In Dolega's case all the synchondroses persisted; and all the epiphyses, the ends of the vertebral arches (spines), and the vertebral (transverse) processes were, in the main, cartilaginous and contained only small ossifying centres. In the bodies the bony centres reached to the periosteum all round, but the epiphysial cartilage plates above and below, and the contiguous swollen discs, protruded beyond them for a considerable part of the circumference, giving the column a somewhat moniliform appearance.

In two of Stoccada's cases (Nos. 2 and 6) the height of each lumbar vertebra was only 15 mm., whereas normally it should be from 25 to 30 mm. On the other hand, the height of the intervertebral disc, which in the normal individual varies from 5 to 7 mm., was 13 mm. Stoccada pointed out the resemblance of this shortening to that which was present in the basis cranii, and concluded that it meant restricted bone formation, whilst the broadening of the discs resulted from cartilaginous proliferation. Incidentally it pointed to the cretinous influence affecting the marrow first, and later on the cartilage. The cartilage proliferation, however, does not make up for the diminished bone formation; consequently in cretins the spinal column as a whole is backward in growth.

THE PELVIS.

The pelvis, again in Dolega's case, is described as being ridiculously diminutive, its true conjugate being two-thirds and its breadth half the normal dimensions. It was like the pelvis of a newly-born infant. The sacro-iliac synchondrosis was present, the three constituent parts of each innominate bone were joined together by cartilage, and the acetabulum was cartilaginous. Indeed, the greater part of the innominate bone was still cartilaginous, and the symphysis blended with it.

THE APPEARANCES IN CACHEXIA STRUMIPRIVA.

It is not easy to find many post-mortem records of cases of cachexia strumipriva in young people, yet, in the days before the value of thyroid treatment had been recognized, the condition cannot have been rare, for Kocher²¹ speaks of the best pupils at school becoming (after the operation) so reduced in mental powers that "the teachers were forced to give up their special interest in them". A full account of one—which is spoken of as Professor Paul Bruns', but which is usually referred to as the Grundler-Nauwork case—is given on pp. 105–109 of the Report of the Clinical Society's Committee on Myxœdema.

The patient, a bright lively boy, had the thyroid removed for goitre at the age of 10 (1866). A marked psychical change took place after the operation, and eighteen years later he was found to have become a dwarfy cretin. Growth in length of the trunk and limbs had ceased after the operation, and only the dimensions of his head were those of a man of his age. The length of his body was the average length of a boy of 10,* and the expression of his face was that of an idiot.

* The average height of a British boy between 11 and 12 years of age is 52 in. (*Report of Anthropometric Committee of the British Association for the Advancement of Science*, p. 31).

He had also the signs and symptoms which characterize myxœdema. He died suddenly during an attack of unconsciousness and arrest of respiration, similar to one from which he had recovered two hours previously.

At the post-mortem "the right humerus, which was removed from the body, showed normal configuration. The border of the epiphysis (was) mostly still recognizable, also that on the proximal end of the femur was still very distinct. The epiphysis of the trochanter major was still cartilaginous."

Wegelin⁴ (on p. 615) relates the case of a man of 47 :—

When 13 years of age he had had a considerable portion of a goitre removed. Up to that time his mental and bodily development had been quite normal, but afterwards growth failed completely, and his intelligence ceased to develop. He could read and write, but could not learn by heart. He was capable of doing work on the land and of making shoes. Death took place in hospital.

Though the thyroidectomy had not been a complete one, he had a cretin's countenance, very little hair in the axillæ and on the pubes, and the body was only 129 cm. long. The basilar synchondrosis and the upper epiphysial disc of the femur were present; there were some remains of the epiphysial cartilage of the upper end of the humerus, and a cartilaginous portion of the innominate bone below the acetabulum persisted. The lower epiphysial disc of the femur had disappeared, but in its place was a band of red marrow contrasting noticeably with the fat-marrow of the rest of the bone.

The microscopic appearances were similar to those in endemic cretinism, but there was a considerable amount of mucoid softening of the cartilaginous intercellular substance of the upper femoral disc.

HISTOLOGY.

The chief features of the histology of cretinism are :—

1. *A diminution in the amount of cartilage proliferation in the ossification zones.*
2. *The formation of a continuous layer of bone separating the proliferating cartilage from the marrow.*
3. *A change in the marrow itself from an active cellular tissue to a more or less inert fatty one.*

These peculiarities are met with both in endemic and sporadic cretinism, and with less certainty in cachexia strumipriva, but variations may depend on the stage which the disease has reached, or on other attendant circumstances.

1. THE DIMINUTION IN THE AMOUNT OF CARTILAGE PROLIFERATION IN THE OSSIFICATION ZONES.

The cartilage cells proliferate on somewhat normal lines, but the depth of the proliferation zone is much less than in the healthy individual. The rows or columns of cells are distinctly shorter, and sometimes broader than normal, and their direction may vary enormously—from the vertical even to the transverse. Instead of columns, rounded groups containing many cells may form, and sometimes attempts at row-formation may be altogether absent, and only a narrow line of enlarged proliferated cells lie along the edge of the cartilage. The picture is essentially one of diminished proliferation, and of a want of orderly arrangement of the proliferated cells. Stoccarda thinks the latter is due to a mechanical cause and gives the following explanation. The cartilage of a synchondrosis, or a cartilaginous epiphysial disc, comes to lie between two roughly parallel layers of bone whose formation

will be described in the next section. As the cartilage tends to grow in depth by proliferation of its cells the internal pressure increases, and the cells are squeezed out of their columnar formation into rounded or oval groups, or may even form a continuous layer of enlarged cells lying up against these abnormal bony layers.



FIG. 312.—Section through the cartilage of the iliac crest from a 57-year-old cretin (Zwygart). Fairly marked cartilage proliferation with irregular cell rows. Bony boundary layer shows only two or three primitive medullary spaces. (After Stoccarda.)

On the other hand, in such parts as the iliac crest or the end of a rib, where the cartilage is not sandwiched between tracts of bone, better proliferation and comparatively well-shaped cell rows occur (Fig. 312). "Calcification of the intercellular substance of the cartilage is always seen in the ossification zone, but its depth depends upon the degree of the cell proliferation. When the latter is fairly normal the calcification zone shows a considerable but not quite normal depth. When proliferation fails it forms only a very small streak, which when slightly magnified appears as a dark blue line close to the bone" (due to the stain).

Rarely the cartilage may show signs of a degenerative change in the shape of small patches of mucoid softening (Fig. 313). These

may be attributed to poor nutrition owing to the impaired vitality of the marrow (*see* p. 391).

2. THE CONTINUOUS LAYER OF BONE SEPARATING THE CARTILAGE FROM THE MEDULLA—THE 'BOUNDARY BONE LAMELLA'.

The most pronounced variation from the normal in the ossification zone in cretins is the presence of a continuous layer of bone between the cartilage and the adjacent cancellous tissue and medulla. In the synchondroses an osseous lamella is present on both sides of the cartilage. In the epiphyses of the long bones one bony layer separates the disc from the diaphysis, and another bounds the ossifying centre, so that the epiphysial discs, like the synchondroses, come to lie between two osseous tracts which are supported upon a cancellous framework (Fig. 319).

This abnormal layer may be spoken of as the 'boundary bone lamella'. It develops from calcified cartilage on the side facing the cartilage, and by the apposition of osteoblasts on the medullary surface, and it may show some signs of lamination. Though more or less continuous it may be interrupted in places by channels through which the marrow invades the calcified zone, or it may show spaces in its substance containing marrow (primitive marrow spaces). Similar spaces, often surrounded by an osseous envelope, may be

seen in the adjacent calcified cartilage. These channels and spaces may, however, be absent from considerable tracts of the lamella. They are more numerous in the younger cretins, and in such show a tendency to penetrate into the cartilage in places; but in older ones they are apt to lie parallel to the bone lamella and to be contained by a bony wall. The contents also vary with age, and show a tendency to become fatty, with very little vascular marrow, in the older cases. The 'boundary lamella' varies in thickness even in the same individual, but there is nothing to show that it grows thicker with age. It may show small excrescences on the side towards the cartilage projecting into the calcified zone (Fig. 308). This is especially the case when the lamella is much broken up by medullary spaces, which is an indication of a more active medulla and a better nourished ossification zone. On its medullary side trabeculae spring from it to blend with the adjacent cancellous structure.

These trabeculae, which may show traces of the inclusion of portions of calcified cartilage, are fewer and often thicker than normal, and consequently the medullary spaces up against the lamella are wider and not so numerous as in ordinary bone. Such is the condition present in endemic cretins of adult age (Stoccada).

A similar boundary lamella occurs in sporadic cases. The illustrations (Figs. 313-316) show the conditions present in the sternum of a sporadic cretin, age 24. A thin layer of bone is shown on both sides of the cartilage which still persists between two adjacent ossific centres of the body.*

FIG. 313.—Section across a synchondrosis uniting two ossific centres in a sternum from a sporadic cretin, age 24, showing the anterior and posterior perichondrium or periosteum, a 'bony boundary lamella' on either side of the cartilage, and patches of degeneration in the central portion of the cartilage. ($\times 5$.) (See text and footnote.)



* R. C. S. Museum, No. 4178.2. I am indebted to the courtesy of Dr. R. M. Stewart, of Leavesden, for the following epitome of the case: "The patient, aged 24, was a perfect example of cretinism; height 3 ft. 2 in.; weight 4 st. 3 lb.; cretinous facies; lips thick; nostrils swollen and everted, remarkably broad and directed forwards; eyelids swollen, with a waxy-like translucent appearance; tongue large, swollen, protruded. Typical elastic swellings above the clavicle. No pubic or axillary hair. Slight umbilical hernia. Testes undescended. Fontanelles closed. Sternum showed delayed ossification. Thymus present, weighed 1 oz. Pituitary and adrenals normal. No vestige of thyroid, but parathyroids could be identified."



FIG. 314.—Enlarged portion of *Fig. 313*. Note the line of enlarged cartilage cells bordering the boundary bone lamella, and the large proportion of fat to cellular marrow. ($\times 12$.)

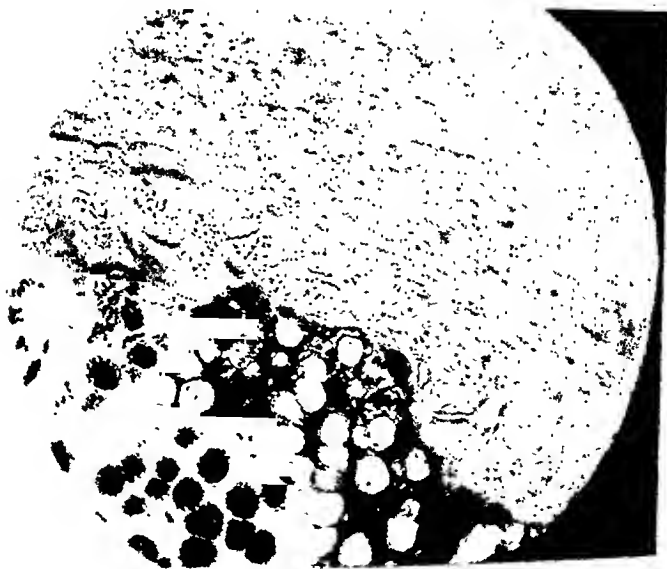


FIG. 315.—A more highly magnified portion of *Fig. 313*, showing the shallow zone of proliferating cartilage cells with calcification of the matrix in immediate proximity to the boundary bone lamella. ($\times 50$.)

In Dieterle's case of a 5-months-old infant, with total absence of the thyroid, the lamella was present in the long bones; whilst in the 14-months Bertha Engel (*see* pp. 391, 392), the conditions present in the basilar synchondrosis pointed to the gradual consolidation of a very pronounced boundary lamella (*Figs.* 317, 318).

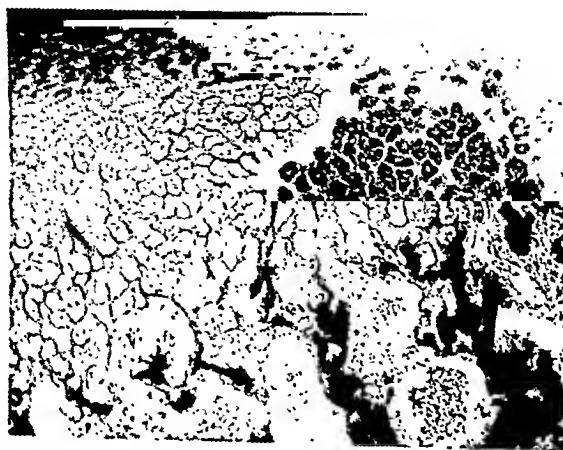


FIG. 315A.—From a normal full-time fetus. Part of the periphery of an ossific nucleus in the body of the sternum, to compare with *Fig.* 315. Note the cartilage proliferation, the advancing medullary spaces, and the absence of fat in the medulla. ($\times 50$.)



FIG. 316.—A still more highly magnified portion of *Fig.* 313, showing the boundary bone lamella and the proliferating cartilage cells, some groups of which are becoming surrounded with calcified matrix developing as protrusions from the bone lamella. ($\times 150$.) (*Figs.* 313-316 by Dr. G. H. Rodman.)

We may assume further that a similar change occurs in cachexia strumipriva, for Stoeccada showed that, in a young rabbit twenty-two days after thyroidectomy, a thin continuous layer of bone developed between the cartilage and the medullary spaces on either side of the spheno-occipital

synchondrosis ; and Dieterle²² found a similar transverse lamella at an epiphysial boundary of a kitten on the seventh day after the thyroid had been removed.

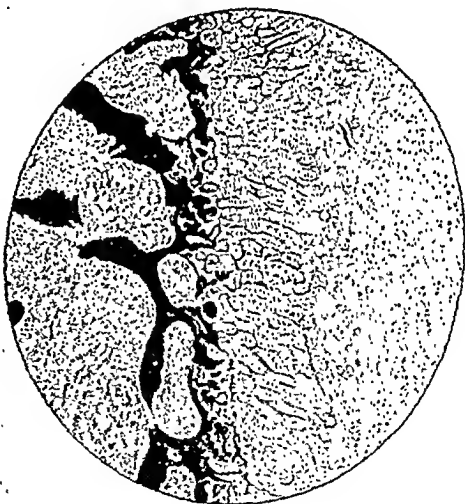


FIG. 317.—Sporadic cretinism. Section through the sphenoid-occipital synchondrosis of Bertha Engel, age 14 months. (*Virchow, Langhans.*) Sphenoid side. In the ossification zone the cartilage cells form short, irregular rows, having various directions. The medullary spaces are less numerous than normal ; almost in every case on the cartilage side they are closed up by bone. (*After Stoccarda.*)

The formation of a bony line of demarcation between the cartilage and the cancellous tissue is the natural consequence of the

termination of ossification when, as at a joint surface, a permanent covering of cartilage is a normal condition. It forms the subchondral compact layer (*see Figs. 71, 74, Inflammatory and Toxic Diseases of Bone*). But such a layer is abnormal in connection with synchondroses and

FIG. 318.—For comparison with Fig. 317. Normal sphenoid-occipital from an 11-months-old girl, occipital side. Very long cartilage cell rows ; numerous medullary spaces. (*After Stoccarda.*)



epiphysial discs, in which the cessation of endochondral ossification is marked by the disappearance of the cartilage and the fusion of the cancellous tissue on either side of it.*

* The presence of a 'boundary lamella', though a fairly constant feature in cretinous bones, is in no sense a diagnostic one. It is seen in a partial form in achondroplasia. There is probably a tendency for it to form in other conditions of ill health when the process of ossification becomes greatly enfeebled. When it does form in such a case the cartilage cells, as in cretinism, show a certain amount of proliferation, but the zone of proliferation is shallow and the degree may be slight. On the other hand, a section through the joint between the manubrium and the body of the sternum in a middle-aged man showed the cartilage limited by a very definite transverse tract of bone of some thickness and without any primitive medullary spaces, but the adjacent cartilage was absolutely quiescent ; there was no cell proliferation, the cell spaces were few, and the cells themselves atrophic.

In two cases there has been described an invasion of fibrous or connective tissue from the periosteum between the epiphysis and the diaphysis like that seen in achondroplasia. In Bowlby's case the thyroid was absent. "The cartilage cells were all about the same size, and were not arranged in rows at the junction of the epiphysis with the shaft; there was no evidence of ossification progressing at this point, and between the bone of the shaft and the cartilaginous epiphysis was a well-marked layer of connective tissue. The condition was constant. The ingrowth of the connective tissue was continuous with the periosteum."

The specimen is in St. Bartholomew's Hospital Museum (No. 3492b). The foetus is a very large one, but the shortness of the limbs, especially noticeable in the arms, which only reach to the level of the umbilicus, and the layer of connective tissue (obvious to the naked eye) at the junction of the epiphyses with the diaphyses in the long bones, as well as the histology, are strongly in favour of achondroplasia, which the absence of synostosis in the 'tribasilar bone' does not necessarily negative. The absence of the thyroid, however, would seem to justify the diagnosis of sporadic cretinism (*see* p. 373).

This ambiguous case is susceptible of a simple explanation; an achondroplasia is born dead and found to have no thyroid. If the child had survived, some signs of cretinism might have made their appearance in the usual course and *at the usual time*, i.e., within the first or second year of life. Perhaps the best description of the case would be "an achondroplastic foetus associated with aplasia of the thyroid".

Dolega's description of the cartilage-cell proliferation and row arrangement in his case tallies with that seen in cretins, but he also gives an account of an ingrowth from the periosteum at the epiphysio-diaphysial junction passing transversely to the long axis of the bone, such as occurs in achondroplasia. In the immediate neighbourhood of this ingrowth there was no row formation, but at its termination rows of cartilage cells appeared approximating to the normal character, "and not extremely meagre as in foetal rickets". Dolega compared this ingrowth to the condition illustrated by Boge,²³ which was evidently taken from a case of achondroplasia.

The histology of this case savours of both cretinism and achondroplasia, and in this difficulty it may be noted that Dr. John Thomson²⁴ speaks of achondroplasia, infantilism, and other kinds of dwarfing as having been described, not rarely, in families in which one or more sporadic cretins have occurred.

3. THE CHANGE IN THE MARROW.

Several observers have drawn attention to an alteration in the marrow of cretinous bones which they consider significant. The ordinary cellular medulla is replaced more or less completely by fat. Langhans,²⁵ who examined the bones of Kocher's 14-months cretin (Bertha Engel) in whom no thyroid was found, states that only in the clavicle had the marrow a partially infantile character, whilst in the humerus, femur, radius, ulna, tibia, and fibula fat-marrow was present throughout.

In the case of the infant of 5 months recorded by Dieterle²⁶ and already mentioned (p. 389), the bone-marrow was remarkable for its rich

fat content. Large quantities of fat cells occurred in the vicinity of the ossification boundary and at the periphery of the shafts, whilst many patches of cellular marrow were seen towards the centre of the diaphysis. This was the opposite of the condition seen in the normal new-born child, in which fat collections are only seen in the older medullary spaces of the diaphysis (i.e., in the central parts) and none can be demonstrated in the neighbourhood of the epiphysis, where the medullary spaces are closely packed with cells.

An excess of fat also is seen in the photomicrograph from the cretin's sternum (*see Fig. 314*) in comparison with that found in sections made from individuals of different ages to contrast with it (*see Fig. 315A*).

Langhans, again, in the examination of 5 adult cretins (ages varying from 26 to 60, and no doubt endemic), found the marrow fatty in the epiphyses and also to a varying extent in the diaphyses, but true marrow tissue was more in evidence than in the infant, Bertha Engel.*

Stoecada, who described 6 examples of endemic cretinism (ages 27, 30,

35, 44, 47, 57), limited his examination in the majority to the sphenoccipital synchondrosis. At this joint the fatty change in the neighbouring medulla was only slight in comparison with that found in the long bones of his second case (Bosshard), in whom the marrow at both ends of the femur was chiefly composed of fat cells and only in a few places were true marrow-cells to be seen (*Fig. 319*), whilst at the upper end of the humerus close to the disc the marrow was composed of fat cells and numerous marrow-cells. He also examined the same synchondrosis in Bertha Engel with a similar result, which contrasted with the observations of Langhans on her other bones (*see Fig. 317*).

The relatively smaller proportion of fat in the neighbourhood

of the basilar synchondrosis when compared with the condition in the long bones does not call for any special explanation, for normally "in the short flat irregular bones, especially in the sternum, ribs, vertebrae, and cranial bones. the red marrow undergoes a much less degree of this fatty change" (i.e., the



FIG. 319.—Section through the epiphysial disc of the upper end of the femur from the same case as *Fig. 308*. Irregular cell rows on the diaphysial side of the cartilago. The bony boundary lamella on both sides shows no medullary spaces. (*After Stoecada.*)

* NOTE ON 'BERTHA ENGEL'.—This case has evidently been the subject of errors which have made it difficult to trace. It is illustrated by Koehler (*Deut. Zeits. f. Chir.*, 1892, xxxiv, 626), and is obviously a girl, whose age is given as 1 year. Langhans gives a full description of the bones and speaks of it as a 14-months-old boy, but refers to Koehler's illustration (*Virchow's Archiv.*, cxlix, pp. 162, 165, 170). Stoecada refers to it as a 14-months individual. gives Langhans' description, and adds his own examination of the basilar synchondrosis, and the name—Bertha Engel. (*Ziegler's Beitr.*, lxi, 492-3.)

change that causes the cellular marrow to disappear from the central part of the shaft as age advances), though even in these situations there is a progressive increase in its amount, which may be very considerable in advanced age and in some pathological conditions.²⁷

Wegelin's statement that the microscopie appearances in *eachexia strumipriva* are similar to those of endemic cretinism has already been mentioned, and in the case he relates (age 47) the fatty condition of the marrow in the femur was brought into prominence by a band of red marrow which marked the site of the vanished epiphysial disc.

Stoecada's experiments on rabbits lend support to these observations; more fat cells were seen in the sections through the basilar synchondrosis of thyroidectomized animals than in the normal marrow of a similar section from a control animal.

These facts show that there is a marked tendency to the 'degradation', as it is termed, of the hæmopoietic marrow in both forms of cretinism, and also in *eachexia strumipriva*. One of the consequences of this change, which results in a great diminution of the amount of functioning blood-forming tissue in the bones, is seen in the anæmia so usually associated with cretinism. Another is the effect on the process of ossification. In this process a healthy vigorous marrow is a very necessary factor. Its aggressive sprouts invade the zone of preliminary calcification, absorb the cartilage, and lay the foundation of the new medullary spaces. This invasion by the marrow is continually going on. But when an actively functioning medulla becomes largely converted to inert fatty tissue its activity must be seriously interfered with, and the length-growth of the long bones consequently more or less interrupted. It is to the inefficiency of the marrow in the invasion and absorption of the cartilage that Langhans, Dieterle, Wegelin, and Stoecada attribute the primary rôle in the disturbance of bone growth in cretinism—and especially in *athyreosis*. The last observer, moreover, looks upon the smaller number of primitive medullary spaces, present even when the medulla shows no great fatty change, and the fact that they are nearly always separated by bone from the cartilage, as a further sign of incapacity on the part of the marrow.*

It has been suggested that the diminished proliferation of the cartilage cells indicates the cartilage as being the seat of primary disturbance, and that the change in the marrow is secondary to it; but Langhans pointed out that in *achondroplasia*, in which proliferation of cartilage cells practically ceases, the marrow has both a normal appearance and activity.

The Presence of Lymphocytes in the Bone-marrow.—In many instances when the marrow has been carefully examined the presence of rounded masses of lymphocytes has been noted. Stoecada states that "the bone-marrow of cretins is distinguished by the presence of groups of lymphocytes or lymph

* It should be noted that though these authorities regard the altered marrow as playing a primary rôle in the disturbance of ossification this does not mean that the cartilage changes as a whole depend upon it. The cretinic influence is, no doubt, injurious to the cartilage and the periosteum as well as to the marrow. The amount of true marrow tissue is often considerable, even when ossification is very markedly delayed, as in the case of the sternum from which certain illustrations (Figs. 313-316) have been made. It is the impaired vitality of the marrow, of which the fatty degeneration is probably an advanced sign, that they recognize as of prime importance.

follicles, and by the increase of eosinophil cells. Probably hypothyroidism is responsible for both." In this connection we may note that Falta quotes McCarrison as finding, in over a hundred blood examinations in endemic goitre, a regular increase in lymphocytes, and in most cases hyper-eosinophilia. Lymphocytes are not normally present in bone-marrow. How then do they come to be there? I am indebted to Dr. A. Piney for the following suggestion. If the collections are found amongst the fat, they will probably be connected with the round-cell infiltration, which is usually found in atrophy; but if they are placed in the cellular marrow, they are probably due to the perversion of the ancestral cell and may be accounted for by the absence of the stimulus that keeps up the activity of the marrow cells.

PATHOGENESIS.

The steps in the evolution of our knowledge of the pathogenesis of cretinism are briefly given by Hector MacKenzie in Allbutt and Rolleston's *System of Medicine* (iv, pt. 1, p. 337); and many interesting details are to be found in the historical and general summaries of the *Report of the Clinical Society's Committee on Myxœdema* (pp. 9 and 177).

In 1873 Gull described "a cretinoid state supervening in adult life in women". In 1877 Ord gave to this state the name 'myxœdema', and noted that the alveoli of the thyroid gland "were compressed and mostly annihilated" by a growth of mucinous interstitial substance. Reverdin in 1882, and Th. Kocher in 1883, described independently a peculiar condition which supervened after total extirpation of the thyroid, to which the latter gave the name 'cachexia strumipriva'. "Sir Felix Semon, struck by the resemblance of the condition described by Kocher to myxœdema, set on foot the investigation of the Clinical Society of London, which brought together a vast amount of evidence tending to prove the identity of the condition with myxœdema." Then Victor Horsley showed that a dyscrasia similar to cachexia strumipriva could be produced in some animals by thyroidectomy, and Schiff found that the ill-effects of that operation could be mitigated by the previous transplantation of a thyroid gland.

Moreover, the resemblance of the mental and bodily changes of cretinism to those that developed in cases of cachexia strumipriva was obvious; and there was also a striking similarity between those changes and the symptoms of myxœdema. Lastly, myxœdema was found to be associated nearly always with an atrophied condition of the thyroid, similar in character to that found in sporadic cretins when the gland was present; but in many sporadic cases the thyroid was absent altogether. These facts pointed to the conclusion that the essential cause of cretinism was to be traced to the loss of the thyroid function; and this was held to be proved when it was shown that the administration of thyroid substance caused the disappearance of the symptoms not only of myxœdema but of cachexia strumipriva and of sporadic cretinism.

The pathological changes in the structure of the thyroid gland are to all intents and purposes similar in myxœdema and in endemic and sporadic cretinism. The atrophy of the gland is brought about by a process of atrophic

cirrhosis, and even when there is a goitre, which is frequently the case in the endemic disease, the secreting structure is affected in this way. The microscopic features presented by the thyroid of a sporadic cretin 7 years old recorded by Packard and Hands²⁸ may be regarded as typical. Briefly put they amounted to this: The alveoli were separated by bands of white fibrous tissue; the epithelial cells were abundant and varied in size; the acini were small, and many contained no colloid substance, and those that did showed it in very small amount. In addition, in this case, the larger blood-vessels of the thyroid, especially the veins around the gland, showed decided calcareous changes.

In the Clinical Society's Myxœdema Report there is an account of the process as it is met with in adult myxœdema. It is stated (p. 44) that "the morbid condition seems to begin as a small-celled infiltration of the walls of the vesicles, and this is accompanied or soon followed by epithelial proliferation in the vesicles themselves. In a more advanced stage the gland becomes converted into a delicate fibrous tissue in which clumps of small round cells, clearly the remains of vesicles, are scattered. In the last stage the gland structure is replaced by fibrous tissue, in which small islets of round cells are sparsely placed."

In endemic cretins there is always some functioning thyroid tissue to be found, notwithstanding the presence of sclerotic and atrophic changes (Falta). The latter are diffused throughout the whole gland and begin in infancy, a fact which is important from its bearing on the delayed growth of the bones (Wegelin). But the degeneration of the thyroid which is met with in cretinism cannot be looked upon as diagnostic, for similar histological changes have been found in cases that were not cretinous (Wordt, 5 cases).

THE CAUSE OF THE CRETINOUS SYMPTOMS.

It is commonly believed that the defective supply of thyroid gland secretion is responsible for the symptoms of endemic and sporadic cretinism, and also of cachexia strumipriva; and, further, that the deficiency may depend on degeneration, congenital absence, or surgical removal of the gland (Kocher and von Wagner). So far as sporadic cretinism and cachexia strumipriva are concerned there is no difference of opinion, but with regard to endemic cretinism there is. Certain observers think that the symptoms of endemic cretinism, such as the mental state, the disturbances of growth, and the degeneration of the thyroid, are separate consequences of a poison—a cretinoid noxa or *materies morbi*—acting directly upon the brain, the bones, and the thyroid gland (Bircher, Ewald, Scholz).

The fact that in athyrosis these symptoms depend upon a failure of thyroid secretion, and that in endemic cretinism thyroid secretion is also deficient, makes the proof of this theory very difficult. Yet it will be seen that there is some justification for it. Falta,²⁹ who inclines to it though not without reservation, gives the following list of factors which are opposed to the hypothesis of a thyroid disturbance alone: "(1) Thyroid therapy is not so consistently useful in endemic as in sporadic cretinism. (2) Myxœdematous symptoms are wanting in many cases, or are only very slightly expressed. (3) There are more variations in the clinical manifestations of endemic cretinism" (than of sporadic). "It is frequently associated with mutism.

There are also cases of mutism which show only few signs of cretinic degeneration. The main features of the cretinous state may be unequally developed; thus cases with brachiocele and high-grade disturbance of growth may be well developed mentally; on the other hand, cases that are highly idiotic may be only slightly behind in growth. (4) There exists a quantitative and qualitative difference in the disturbance of growth. This is only delayed in endemic cretinism; even in the highest grades there occurs late closure of most of the epiphysal junctures; the disturbance in growth is disproportionate.* (5) The hypophysis is often goitrously degenerated.†

FACTS POINTING TO A TOXIC ORIGIN OF THE DISEASE.

Valuable information on the subject of a possible toxic (noxa) origin of the changes which are thought to lead to defective thyroid secretion has been furnished by the researches of Colonel R. McCarrison. That officer made many investigations as to the etiology of endemic goitre and cretinism in India, and came to the conclusions: (1) that the main cause of endemic goitre is to be traced to the excreta of human and animal subjects; (2) that the infecting agent is capable of life, and possibly of some degree of growth, outside the body in contaminated soil or grossly polluted water; and (3) that water, food, soil, etc., by which the infecting agent reaches the body, are of importance only as vehicles of transmission. He found no evidence that the blood or the thyroid glands of goitrous individuals contain bacterial or protozoal organisms, but, rather, reason to think that intestinal anaerobes possess a goitrogenous influence on the thyroid, and that the toxic agent of endemic goitre is produced in the intestinal tract of man and animals.³⁰ One of the most interesting of McCarrison's observations was the following:—

Goitre is very common in the village of Kashrote (near Gilgit), and the water-supply is contaminated as it flows in open channels through several other villages higher up the valley. Water, which had previously been made muddy by agitation whilst flowing through its channel, was brought daily from Kashrote, and filtered in large quantities through a Berkefeld house filter. The deposit on the candle was washed off in distilled water, and a quantity of the dark grey mixture, equal to about 4 oz., was given in milk every morning before the first meal of the day to six healthy young men (18 to 20 years) who were carefully protected from infection from other sources. Two of these, who were regarded as susceptible because they had previously suffered from small enlargements of the thyroid which had recovered when they left Gilgit, developed a swelling of the thyroid, which made its appearance in about a fortnight. McCarrison submitted himself to the same experiment, and developed a swollen thyroid on the fifteenth day. Similar experiments were repeated, and on

* He refers here to Breus and Kolisko, who state that in six cretin skeletons which they examined, "never did all the epiphysal junctures remain open until an advanced age; therefore there did not exist in all bones the same degree of disturbance of growth, and that there resulted from this a disproportioned skeleton".

† Schönmänn's investigations show that, where goitre is endemic, strumous alterations are found very commonly in the glandular part of the hypophysis. "In persons with struma of the thyroid there was always found enlargement of the hypophysis and proliferation of the connective stroma, also chromophilic strumas or strumas with especially vascular development of the stroma and hyaline degeneration and swelling of the columns of cells, and finally those with marked colloid formation. Cyon found strumous alterations of the hypophysis very frequently in Bernese dogs. The goitre poison therefore works deleteriously on the hypophysis" (Falta, loc. cit., 164).

each occasion some of the subjects developed a thyroid enlargement. On the other hand, no effect was produced in any of the control cases who were given the same suspended filtrate after it had been boiled.³¹

In the next section of his book³¹ (p. 94) he discusses E. Bircher's experiments on rats, which substantiate his own findings in man; and both observers agree that "the presence in suspension in goitre-producing waters of a living agent, which is the direct or indirect cause of the disease, has been demonstrated."

Another experiment had a closer relation to cretinism. Two presumably healthy rats were fed on the filtrate of an emulsion of faeces from a goitrous person. At the termination of the experiment both animals had contracted goitres, the mother's being considerably larger than the father's. Of 31 animals born from these rats in six litters during the experiment, the first litter of 6 were born too soon for changes to have been expected, and were normal; the last litter of 5 were all consumed by the mother in the first twenty-four hours. Of the remaining 20, 16 were examined: 3 were cretins; 7 had congenital goitre, 2 of which showed partial fibrosis of the thyroid; 7 were normal; whilst changes in the parathyroids of greater or lesser degree were present in 5.³²

SUMMARY.

The present position of the pathogenesis may be briefly stated as follows.

The essential cause of the symptoms of cretinism, myxœdema, and cachexia strumipriva is an absence or failure of the thyroid gland secretion. In sporadic cretins, when the thyroid is absent, the failure is due to congenital defect; in cachexia strumipriva to the surgical removal of the whole gland.

In other cases—viz., those sporadic cases in which a thyroid is present, endemic cretins with or without goitre, and myxœdema—a gradual suppression of the secretion results as a consequence of atrophic degeneration of the gland tissues. The cause of this degeneration is not definitely settled, but the researches of McCarrison and Bircher point strongly to a toxic agent of bacterial or protozoal origin arising in connection with human or animal faeces.

In the endemic disease the living agent is probably conveyed to the individual by contaminated food or water. In sporadic cases with thyroids, and in myxœdema, the presumption is that the toxic substances may arise from the patient's natural intestinal micro-organisms. The thyroid in the sporadic cases is often the subject of some mal-development, which would seem to suggest its feebleness, and consequently greater susceptibility to toxic irritation. Finally, the change in the gland is of an inflammatory nature—a point in favour of the toxic theory, and opposed to the idea of a derangement of hormone influence.

TREATMENT.

The essential treatment of cretinism consists in the administration of thyroid extract, but its use must be continued during the whole lifetime of the individual, for relapse follows its cessation. The results of thyroid therapy in the two varieties of cretinism are not equally satisfactory. In *sporadic cretinism* the effect is marvellous. The swollen, œdematous-looking

condition of the skin and face gradually subsides, "the large protruded tongue recedes behind the teeth", and the supraclavicular swellings disappear. The deformity of the head and the flattened bridge of the nose grow less marked, and "the over-due teeth begin to appear". The lumbar lordosis and the pendulous abdomen assume a more natural contour; the umbilical hernia often vanishes; the testes descend into the scrotum, and the child begins to walk and run about.

"The remarkable change in the bodily condition extends to the intelligence". The hitherto listless and apathetic infant begins to notice, learns to talk and to play, and acquires a bright and happy countenance. If the condition is recognized early and treatment begun at once, the general improvement is so steady that the sufferer may become practically a normal individual capable of being educated, and of marrying and begetting a family.

In addition to this there occurs a rapid increase of stature and physical development. The quickness and the amount of growth, especially in young children, is perhaps the most striking result of the treatment. Hector Mackenzie says that it is not unusual for a cretin to grow several inches in six months; that the rate of growth "is greatest in the early months of treatment, and that children grow more than adolescents, and adolescents than adults. A young cretin may grow 5 or 6 inches in the first year, 4 or 5 in the second, and then continue to increase in height at the normal rate." The epiphysial lines in due course become ossified.

The influence of thyroid treatment upon stature depends upon the growth in length of the bones—especially of the long bones. It may be illustrated by a few examples. In one case,³³ age 12 years, there was an increase of 2 inches in height and the anterior fontanelle closed after 8 months' treatment. In another²⁸ a boy of 6 years grew 9 cm. in 10 months, and a previously wide open anterior fontanelle entirely closed. In a third³⁴ a female child of 2 years grew from 24 to 32 inches in less than 4 months.

Even in sporadic cases of mature age growth may be started by thyroid treatment, but in some, where the athyreosis is incomplete, the epiphysial lines may have disappeared, and in that case no effect on the stature will be produced. Such a case occurred to Magnus-Levy (Falta); the patient was 45 years old and 132 cm. high; all the junctures were ossified and no increase of growth took place.

The closure of the fontanelle under treatment might at first sight be thought to be due to the stimulation of membranous ossification. To some extent no doubt it is; for though, in cretinism, the cessation of length-growth completely overshadows any deficiency in the formation of the diaphysial cortex, yet periosteal ossification is almost certainly affected as well as endochondral, and the inefficiency of the marrow as shown by its fatty change must necessarily influence both. But another factor probably comes into play. The rapid growth of the cartilaginous base, by increasing the accommodation for the brain, will permit the bones of the vault to come more readily together.

In *endemic cretinism* thyroid therapy shows to the best advantage in the promotion of growth, but it is more uniformly efficacious in the sporadic variety; for in the latter, in the absence of treatment, ossification is at a

standstill, and the epiphysial discs often persist throughout life, whilst in the endemic disease ossification is only delayed, and after 30 years of age the epiphysial junctures and the synchondroses are only exceptionally found open (Bircher). Still ossification is accelerated, stature is increased, and the fontanelles tend to close.

The earlier the treatment is begun, the more satisfactory are the results, and the best are obtained in those cases in which the symptoms of cretinism come on in the first years of life. Falta mentions a case of Wagner's—a boy of 15, who after four years' treatment had grown from 105 cm. to 148 cm., or 29 cm. more than the average growth at that period of life.

The effect on the mental condition, however, is slight, and very different from that in sporadic cases. There is a decrease in the apathy, but in many of the severe cases intelligence and speech are not influenced. It is in the slighter cases that benefit may be hoped for, and in some indeed a cure may follow, and occasionally no relapse takes place, even when the treatment is discontinued. In later life, too, such cases may improve.

In the severe forms of endemic cretinism, however, thyroid treatment is not satisfactory, and it is stated that cases which develop the affection so early that congenital cretinism may be assumed 'are mostly refractory'.

It has been pointed out by several authors that the rapid increase of growth which takes place during thyroid treatment tends to produce a softened condition of the bones. The bones of the lower limbs, which have to bear the weight of the body, are consequently apt to bend if cretins undergoing treatment are allowed to run about and to be continually on their legs. At such times careful watch should be kept upon the rate of growth, and when this is abnormally rapid the patients should be kept in bed, and any tendency to bend corrected by suitable splinting.

I desire to acknowledge gratefully the kind help received from many friends in the preparation of this article. Some of them are mentioned in the appropriate place in the text, but amongst those who could not be referred to there, Sir Arthur Keith, Sir James Berry, Mr. C. F. Beadles, Dr. Lawrence, Dr. G. H. Rodman, Col. M. H. Knaggs, and Miss Glascock, have placed me under special obligation. More particularly am I indebted to Dr. Fabio Stoccada, of Castiglione-Stiviere, for allowing me to reproduce some of the illustrations in his article in *Zeigler's Beiträge*, 1915, lxi. Without them, owing to the difficulty of obtaining the necessary pathological material in this country, it would have been impossible for me to have given an adequate representation of the histology of cretinism.

REFERENCES.

- ¹ CREW'S Researches, *Proc. Roy. Soc.*, Series B, 0924, xcv, 228.
- ² KOCHER, "Zur Verhütung des Cretinismus und cretinoider Zustände nach neuen Forschungen", *Deut. Zeits. f. Chir.*, xxxiv, 605.
- ³ FALTA, *The Ductless Glandular Diseases*, Meyer's trans., 138.
- ⁴ WEGULIN, *Cor.-Blatt. f. schweiz. Aerzte*, 1916, xlv, 611.
- ⁵ PAGGE, HILTON, *Med.-Chir. Trans.*, 1871, liv, 164.
- ⁶ DOLEGA, *Zeigler's Beitr. z. pathol. Anat.*, 1891, ix, 489.

- ⁷ CURLING, *Med.-Chir. Trans.*, xxxiii, 303.
- ⁸ BEACH, FLETCHER, *Trans. Pathol. Soc.*, 1873-4, xxv, 265.
- ⁹ FALTA, *loc. cit.*, 154.
- ¹⁰ FODERE, *Traité du Goitre et du Crétinisme*, Sect. Ixi, 122.
- ¹¹ MACKENZIE, HECTOR, *Allbutt and Rolleston's System of Medicine*, iv, pt. 1, 336.
- ¹² TOWNSEND, C. W., *Arch. of Pediatrics*, 1892, ix, 827.
- ¹³ PHILLIPS, SIDNEY, *Clin. Soc. Trans.*, 1885, xviii, 253.
- ¹⁴ FALTA, *loc. cit.*, 159.
- ¹⁵ BIRCHER, H., *Vollmann's Klin. Vortrüg. Chirurg.*, 1890, 3393; *Jahrb. f. Kinderheilk.*, 1906, lxiv, 588.
- ¹⁶ *Report of Clinical Soc. Committee on Myxædema*, 46.
- ¹⁷ HORSLEY, VICTOR, "Cretinism", *Tuke's Dictionary of Psychological Medicine*.
- ¹⁸ HERMAN, N. Y. *State Jour. of Med.*, 1914, Aug.; *Arch. of Pediatrics*, 1917, xxxiv, 831.
- ¹⁹ WEGELIN, C., *Cor.-Blatt. f. schweiz. Aerzte*, 1916, xlvi, 614.
- ²⁰ DOLEGA, "Ein Fall von Cretinismus beruhend auf einer primären Hemmung des Knochenwachstums, etc.", *Zeigler's Beitr. z. pathol. Anat.*, 1891, ix, 488.
- ²¹ *Rep. of Clinical Soc. Committee on Myxædema*, 90.
- ²² DIETERLE, *Virchow's Arch.*, clxxxiv, 93.
- ²³ BOGE, *Ibid.*, 1883, xciii, 442, Taf. ix.
- ²⁴ THOMSON, JOHN, *Clinical Study and Treatment of Young Children*.
- ²⁵ LANGHANS, *Virchow's Arch.*, cxlix.
- ²⁶ DIETERLE, *Ibid.*, clxxxiv, 56.
- ²⁷ BEATTIE and DICKSON, *Textbook of Pathology*, 3rd ed., 583.
- ²⁸ PACKARD and HANDS, *Amer. Jour. Med. Sci.*, 1901, exxii, 289.
- ²⁹ FALTA, *loc. cit.*, 169.
- ³⁰ McCARRISON, R., *Ind. Jour. Med. Research*, 1913-14, i, No. 3; 1914-15, ii, No. 1.
- ³¹ McCARRISON, R., *Etiology of Endemic Goitre*, 88 to 99.
- ³² McCARRISON, R., *Ind. Jour. Med. Research*, 1913-14, i, 516.
- ³³ GEORGE, W. H., *Brit. Med. Jour.*, 1896, ii, 646.
- ³⁴ NOYES, N. Y. *Med. Jour.*, 1896, March 14, 234.

THE EFFECT OF FLAVINE ANTISEPTICS ON TISSUE GROWTH IN VIVO.*

By J. W. S. BLACKLOCK,

PATHOLOGIST AT THE ROYAL HOSPITAL FOR SICK CHILDREN, GLASGOW.

IN a paper published in 1922 by Bennett, Blacklock, and Browning¹ it was shown that compounds of diamino-acridine (flavine group of antiseptics) had no adverse effects on the process of healing as observed clinically, and in material taken for histological examination from various septic lesions (burns of third degree, carbuncle, cellulitis, and osteomyelitis).

However, widely divergent conclusions regarding the effects of the flavine compounds on the process of healing have been reached by various observers. Bashford, Hartley, and Morrison² found that the process (*viz.*, healing under flavine) differs in no essential feature from the production of an eschar by a destructive agent not liquefying the tissues, and that the reaction of repair is removed into the depths and tends to involve the muscles, ultimately leading to a great over-production of scar tissue.

Drummond and McNec³ have stated that flavine cannot be classed as a success in the treatment of the later stages of war wounds, since the wounds tend to assume a stagnant condition when the process of repair is in abeyance. Nevertheless, they found it possible to perform primary suture successfully in wounds even though organisms were present, and also secondary suture in wounds which were far from being bacteriologically sterile.

On the other hand, our findings referred to above were based on over six hundred severe burns and pyogenic conditions met with in hospital wards and treated continuously (in some cases of extensive burns for nearly five months) with 1-1000 solutions of proflavine or acriflavine. Carslaw and Templeton⁴ had previously reported very similar results to ours. Lawson⁵ also found proflavine of great service in ophthalmological work, and states that solutions of this dye are invaluable as a dressing for grafts of all descriptions—*e.g.*, Thiersch's grafts, which, after being dressed with flavine, are not interfered with for at least a week. In a more recent paper⁶ this author states that, since using solutions of flavine regularly for the last ten years, he has not had a single case of septic sutures after strabismus operations, and further he has found flavine "an efficient antiseptic which can, without any irritation whatever, be applied to the wound area, is most helpful, and gives the surgeon a great feeling of security."

On account of the divergence of opinions it seemed of importance to

* From the Pathological Departments of the University and of the Royal Hospital for Sick Children, Glasgow. This work was done in continuation of the investigation previously reported by Bennett, Blacklock, and Browning, with the support of the Medical Research Council.

study further the occurrence of mitosis in the histological material already reported on, as this feature affords invaluable evidence regarding the action of the drug on the growing tissues. Special attention has been paid to the occurrence of mitotic figures in the various types of cells and the actual depth at which these were met with under the wound surface to which the application of flavine was being made.

As regards the mode of application, clinical results, and general histological findings, these have already been fully described. For the present purpose sections were made from the tissues which had been fixed in Zenker's fluid immediately after excision and stained by various methods to show mitotic figures, which were looked for in a zone 0.2 mm. in depth immediately below the actual growing surface of the wound.

In the twelve cases studied mitotic figures were not difficult to find in this zone, being noted at an average distance

of 0.11 mm. below the surface in various types of cells—endothelial, epithelial, large mononuclears, and fibroblasts.

Mitosis was observed in one instance in an endothelial cell in the wall of a small capillary 0.1056 mm. below the surface (*Fig. 320*), this finding being directly opposed to the view expressed by Bashford, Hartley, and Morrison² that the flavine antiseptics kill successive layers of the reacting tissue elements, including the essential vascular mechanism; while on the other hand it explains what has been noted clinically, namely, the slight oozing of blood which often occurs from the surface of a wound when the dressing is removed, owing to damage caused by the gauze adhering slightly to the surface closely under which (often only 0.1 mm.) are the actively growing capillary loops.

It has been clinically observed by Lawson⁵ and ourselves that solutions of the flavine compounds have no deleterious influence on the growth of epidermal or whole-skin grafts, and these findings are further borne out in



FIG. 320.—A mitotic figure is present in an endothelial cell in the wall of a capillary vessel 0.1056 mm. below the wound surface, which is at the top of the figure. (Note: the dark stained material above the actual growing surface of the wound is due to freshly exuded blood-cells and fibrin which escaped during excision of the specimen; this also applies to *Figs. 321 and 322.*) Iron hæmatoxylin. ($\times 350$.)



FIG. 321.—The field at a point where the young epithelium is growing over the wound surface and shows mitosis in an epithelial cell 0.0176 mm. below the growing surface. Iron hæmatoxylin. ($\times 350$.)

the present investigation by the observation of mitosis in young epithelial cells close to the healing margin immediately (0.0176 mm.) under the surface (*Fig. 321*). In the young supporting tissues (fibroblasts, etc.) all stages of mitosis were noted at distances varying from 0.0176 mm. (*Fig. 322*) and upwards below the surface, a finding which is in accord with the observations of Mueller,⁷ who, while studying the growth of tissue cells under the highly artificial conditions of tissue culture *in vitro*, found that the flavine antiseptics were the only substances which did not inhibit cell division in a concentration which was effective against the organisms tested (streptococci).

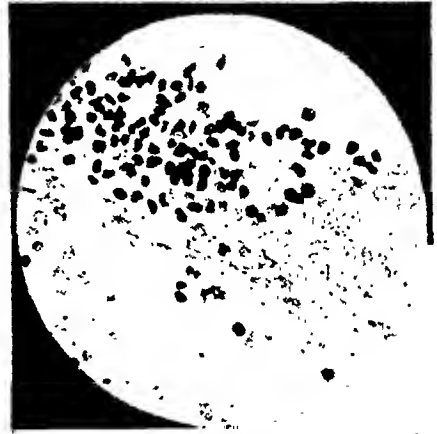


FIG. 322.—Mitosis in a large mononuclear cell, probably a fibroblast, 0.0176 mm. below the growing surface. Iron hæmatoxylin. ($\times 360$.)

It is noteworthy that in the present investigation the dividing nuclei in cells other than endothelial were not in immediate relation to capillary blood-channels, and the type of lesion had no influence on the occurrence of mitotic figures, as these were found, for example, the same distance (0.02 mm.) below the surface in a carbuncle and in a third-degree burn.

Similarly, the length of treatment with flavine solutions, and also the age of the patient, did not affect the distribution of nuclear divisions.

Further, as already observed in our former paper, from the clinical standpoint no interference is found in the growth of bone in cases of osteomyelitis, and this is supported by additional histological observations. For example, in acute suppurative osteomyelitis of the femur in a child, age 10, abundant staphylococci were found in direct films in the material removed at operation; a gutter was cut in the bone which was packed with gauze soaked in 1-1000 neutral acriflavine. The dressing was removed at the end of three days, the gutter and surrounding tissue being thoroughly swabbed with the flavine solution, then re-packed with a similar dressing, which was removed three days later, i.e., six days from commencement of treatment. In a small piece of bone removed for pathological examination at this time, active growth of bone was noted 0.11 mm. under the

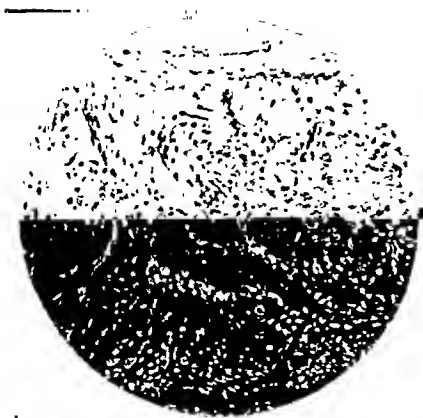


FIG. 323.—Active growth of young bony lamellæ 0.11 mm. under the surface. Iron hæmatoxylin. ($\times 300$.)

surface (*Fig. 323*), and cultures remained sterile when two small pieces (peasized) of bone were inoculated each into 20 c.c. of tryptic broth and incubated for seventy-two hours.

SUMMARY.

Antiseptics of the diamino-acridine series (flavines) do not inhibit the growth of tissue *in vivo* as is shown by the occurrence of mitosis in various types of cells 0.018 to 0.2 mm. below the actual growing surface of the wound.

REFERENCES.

- ¹ BENNETT, C., BLACKLOCK, J. W. S., BROWNING, C. H., *Brit. Med. Jour.*, 1922, ii, 306.
- ² BASHFORD, E. F., HARTLEY, J. N. J., MORRISON, J. T., *Ibid.*, 1917, ii, 849.
- ³ DRUMMOND, H., and MCNEE, J. W., *Lancet*, 1917, ii, 640.
- ⁴ CARSLAW, R. B., and TEMPLETON, W., *Ibid.*, 1918, i, 634.
- ⁵ LAWSON, A., *Ibid.*, 1919, i, 1112.
- ⁶ LAWSON, A., *Brit. Med. Jour.*, 1927, ii, 1128.
- ⁷ MUELLER, J. H., *Jour. Pathol. and Bacteriol.*, 1919, xxii, 308.

AN UNDESCRIBED DISEASE OF BONE.

(Being the substance of a Hunterian Lecture delivered at the Royal College of Surgeons of England on Feb. 10, 1928.)

By J. H. SHELDON,

HON. PHYSICIAN TO THE WOLVERHAMPTON AND STAFFORDSHIRE HOSPITAL.

This paper describes a case which presented a collection of pathological features that are believed to represent a new clinical entity. The specimens are in the Museum of the Royal College of Surgeons, London.

HISTORY.

The patient, a male, was healthy till the age of 11, when he discovered by chance a hard lump on the front of the right knee. This was excised at the Wolverhampton and Staffordshire Hospital, and was found to be an osteoma attached to the head of the tibia. In the operation notes it is stated that the patellar tendon was studded with what appeared to be multiple fibromata. Four years later the lump recurred, but the patient was able to continue at work for a further two years, after which the operation had to be repeated, as the growth was interfering with movement.



FIG. 324.—Right knee in 1922.



FIG. 325.—Left knee in 1922.

During these two years the left knee had also become affected. The state of the knees at the age of 17—before the second operation—is shown in *Figs. 324 and 325*. In the right knee (*Fig. 324*) a large mass of bone of composite nature has developed in the patellar ligament and the capsule of the knee-joint, but is not attached to the tibia. Above the patella there is an area of ossification in the rectus muscle. The outline of the tibia is abnormal, the bone between the condylar surface and the tuberosity having a step-like

appearance, as if there had been some absorption of bone in this region. *Fig. 325*, which illustrates the left knee, is important as it is the earliest record we possess of the manner in which the condition began. There is a line of ossification in the patellar ligament, continuous with the patella but separate from the tibia, the shape of which is normal. Almost immediately



Fig. 326.—General appearance of the patient in October, 1925.

after the second operation on the right knee the disease became more active, the growths at the left knee enlarging and those at the right knee recurring. At about the age of 18 an infective element appeared in the right arm. At the wrist a soft painless swelling formed, which burst, with the discharge of thick yellow pus. The bone became affected and a sinus formed, penetrating the lower end of the ulna and persisting till death. From this there was daily a small amount of cheesy discharge, which was insufficient for animal inoculation, but was repeatedly sterile on culture. Films of the discharge showed pus cells without organisms, and tubercle bacilli were never found. Similar painless swellings appeared on the right elbow, the right arm, and in the right axilla, but after bursting they healed up completely.

In May, 1925, the right shoulder began to enlarge, and beyond a steady increase in its size there were no significant changes until September, 1925, when the patient first came under my observation. In November, 1925, he was shown at the Clinical Section of the Royal Society of Medicine, and a description of the clinical features at that time will be found in the Society's *Proceedings*.¹

The general appearance of the patient is shown in *Fig. 326*. He was a tall man, the measured height being 6 ft. 0½ in., though the height obtained from the lengths of the femur and tibia by Professor Karl Pearson's formula is 6 ft. 3 in., the difference

being due to the ankylosis of the knees in partial flexion.

Figs. 327–329 illustrate the state of the knee-joints at the age of twenty. *Fig. 327* shows the upper part of the right knee, with numerous deposits of bone in the quadriceps muscle and in the capsule of the joint. There is a well-marked transverse striation of the lower end of the femur. *Fig. 328* shows a mass of bone developed in the patellar tendon, having acquired a bony union with the tibia. *Fig. 329* represents the condition of the left knee-joint, and reveals the progress, unhindered by operation, that was made in the three years since the skiagram shown in *Fig. 325* was taken. There is a single plate of bone above the patella, while below it is an enormous mass of composite bone reaching to the tibia. It is united with the tibia close to the tuberosity, but elsewhere there is a clear line of demarcation. Destruction of the bone has led to the anterior part of the tibia having a

semicircular outline. *Fig. 330*, taken after removal of the soft tissues, is added to indicate the progress made in the four months before death, and it is clear that the mass has acquired a more extensive bony union with the tibia.



FIG. 327.—Right knee in 1925. Striation of the lower end of the femur is marked with an arrow.



FIG. 328.—Right knee in 1925.

Figs. 326 and 331 illustrate the progress of the growth at the right shoulder. *Fig. 326* was taken in October, 1925, and at this time the shoulder was occupied by a rounded swelling with a circumference of 20 in., painful, tense, and in places fluctuant. It was considered to be a sarcoma. *Fig. 331*



FIG. 329.—Left knee in 1925. The arrows show how the condition has progressed.



FIG. 330.—Left tibia. The arrow marks bone developed in the peroneus longus muscle.

was taken after death, and shows the enormous size that the tumour ultimately attained. It penetrated under the skin of the chest and back, with the formation of deep pockets in which sepsis inevitably occurred. At the last the mass became so heavy and bulky that movement of the patient was dangerous. Coley's fluid was tried without the slightest effect.

The urine was normal, and albumosuria was never found. The Wassermann reaction was negative. The temperature was consistently irregular, fluctuating between 99° and 101° , due in all probability to the sinus at the wrist. Blood culture was negative.

Blood-count on Oct. 13, 1925: red cells, 5,200,000 per c.mm.; Hb, 70 per cent; colour index, 0.67; white cells, 8,350 per c.mm., with a differential count of polymorphs 37 per cent, lymphocytes 56 per cent, monocytes 3 per cent, eosinophils 1 per cent, basophils 1 per cent. Blood-count on Nov. 2, 1925: red cells, 5,020,000; Hb, 58 per cent; colour index, 0.58; white cells, 12,350, with a differential



FIG. 331.—A post-mortem photograph showing the condition of the shoulder in February, 1926.

count of polymorphs 69 per cent, lymphocytes 25 per cent, monocytes 2 per cent, eosinophils 1 per cent, basophils 1 per cent.

Death occurred from exhaustion on Feb. 14, 1926.

BONY CHANGES IN THE PATIENT'S FATHER.

With the exception of the patient's father, the family history revealed nothing unusual. The father is the subject of a curious set of bony changes. Bony prominences are found at the wrist and elbow on both sides, which have been present for as long as he can remember. They have shown no tendency to enlargement, except for those at the left elbow in consequence of an accident a few years ago. Fig. 332 shows a skiagram of the right elbow. Bony outgrowths can be seen at the lower end of the humerus and at the upper ends of the radius and ulna. Their nature is doubtful, but their position at the epiphysial ends precludes their being due to diaphysial aclasis. Further, there are no other signs of this disease, and the growth of the long bones is not stunted. They probably represent ossifications in the ligaments and capsule of the joint of a similar type to those found so extensively in the son. The presence of such lesions in the father suggests that the disease may have an hereditary basis.



FIG. 332.—Right elbow of patient's father.

POST-MORTEM FINDINGS.

Right Shoulder.—The region of the right shoulder was occupied by the enormous tumour already referred to. Posteriorly it extended to the vertebral column, almost completely enclosing the scapula, and in front it lay along the outer half of the clavicle and the corresponding part of the upper four ribs. On the arm it reached as far as the elbow. It arose from the upper end of the humerus, and weighed 23 lb. 5 oz. The peripheral half was composed of firm white tissue containing numerous cysts, some of which were filled with old altered blood, others with a greenish glairy fluid. In the middle of the growth cartilage was found, which was replaced by bony spicules as the humerus was approached.

Histologically the growth proved to be a spindle-celled osteosarcoma. The peripheral part consisted entirely of spindle cells, which were fairly regular in size and shape. No giant cells were found, and this part of the tumour showed no bone formation. Sections from the bony part near the humerus showed that the spindle cells of the peripheral portion had been to a very large extent replaced by cartilage, and it was in this cartilage that ossification was taking place, the process resulting in the formation of irregular branching trabeculae of osteoid tissue. With the exception of two small plaques of tissue, the size of a waistcoat button, on the costal surface of each lung, no metastases were found. Histologically these consisted of masses of cartilage cells surrounded by spindle cells of the same type as those occurring in the unossified part of the sarcoma of the humerus. The cartilage cells showed no evidence of ossification, but a few very small masses of osseous tissue were present in the spindle cells.

Right Humerus.—This bone was completely embedded in the growth, which could, however, be separated easily from its lower half. The upper half was expanded into a circular mass infiltrated by strands of fibrous tissue, the outer surface of the mass shading off into the area of bony spicules found at the centre of the growth. The lower half of the shaft was normal, except for the presence of an irregular bony ridge arising from the outer margin, and reaching from just below the tumour to the lateral epicondyle. A window cut in this ridge showed that the new bone had been deposited on the outside of the humerus, the normal compact bone of which could be distinguished underlying the cancellous bone of which the ridge was composed. It probably represents a great exaggeration of the lateral epicondylar ridge, formed by ossification in the attachments of the brachioradialis and extensor carpi radialis longus muscles. On longitudinal section of the humerus it was seen that immediately below the sarcoma the medullary cavity was completely blocked by a layer of hard compact bone, while below this again the medullary cavity was filled with aplastic fatty marrow. These changes can be seen in *Fig. 333*.



FIG. 333.
Right humerus.

Right Scapula.—This bone was almost completely embedded in the sarcoma. There is extensive destruction of the glenoid cavity, and a good deal of irregular new formation of bone intermingled with areas of erosion along the axillary margin.

Right Clavicle.—The whole surface of the bone is the subject of much new bony formation, which is pitted and irregular in shape, and cannot be separated cleanly from the fibrous tissue around it. It completely envelops the clavicle, and a transverse section shows a clear line of demarcation between

this new bone and the compact bone of the clavicle. This bony investment reaches a maximum over the area of attachment of the deltoid muscle and over that of the costo-clavicular ligament. The condition is illustrated in *Fig. 334*. The separation of the new bone is due to the manipulation involved in preparing the section, but places can be seen where the trabeculae are still continuous with the parent bone. The new bone consists of a complicated series of trabeculae contained within a layer of fibrous tissue resembling



FIG. 334.—Decalcified section of right clavicle, showing investment of new bone surrounding the clavicle.

periosteum, the appearance suggesting that the new bone has arisen within the periosteum of the clavicle. Enclosed in the meshes of the trabeculae is a thin fibrous tissue containing occasional fat-cells.

Bones of the Right Fore-arm.—The forearm was fixed during life in semipronation, owing to the changes that occurred at the lower end. The *ulna* is normal in its upper half. At its lower end it is firmly united to the radius, owing to the interlocking of nodular masses of bone formed in the interosseous membrane. These have a true bony continuity with the radius, but are everywhere separated from the ulna by fibrous tissue.

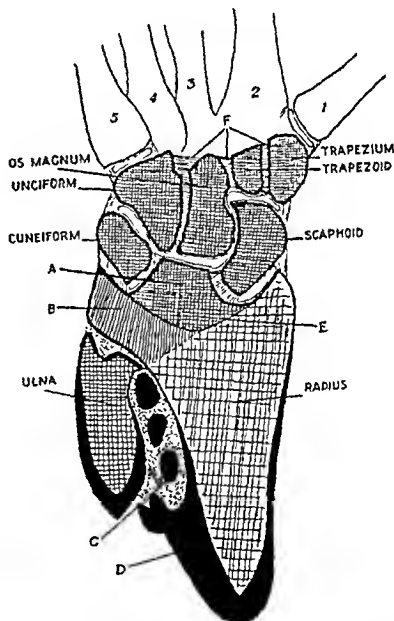


FIG. 335.—Tracing made from the bones of the wrist-joint, as seen in coronal section. A, Semilunar bone, united with the lower end of the radius. The line of junction is marked by E. B, Formation of bone in the articular disc and internal lateral ligament, united by fibrous tissue to the semilunar bone and the radius. C, Nodular exostoses in the interosseous membrane, connected with D. D, Growth of bone from the radius into the interosseous membrane. F, Fibrous union between all the bones of the distal row of the carpus.

The sinus found clinically at the wrist is represented by a hole 4 cm. above the styloid process extending into the centre of the ulna. In the fresh state this was filled with thick white pus.

The *radius* presents an exostosis 8 cm. below the head, 1.5 cm. long and 0.6 cm. high. A section through this shows it to be a true cancellous osteoma, having a core of cancellous bone covered by a layer of compact bone, both of which are continuous with the corresponding parts of the radius. The state of affairs at the lower end of the bone and in the carpus is seen in *Fig. 335*, which is a tracing made from a coronal section through this region: (1) Arising from the lower 11 cm. of the interosseous border of the radius there is an extensive formation of new bone in the interosseous membrane which is almost entirely composed of hard compact bone, of nodular shape. It indents the ulna in places, but is everywhere separated from this bone by fibrous tissue. (2) The lower radio-ulnar joint, including the recessus saciformis, has been obliterated by this new formation. (3) There has been a good deal of absorption of both the compact and cancellous bone at the lower end of the radius, and the marrow is white and appears very unhealthy. (4) The lower end of the radius has become greatly enlarged by the addition to it of a mass of new cancellous bone on its medial side. This includes two distinct elements: (a) There has been a process of ossification which, starting from the medial extremity of the radius, has replaced the whole of the articular disc, and extended thence distally along the internal lateral ligament of the wrist-joint. This, like the formation in the interosseous membrane, is separated from the ulna by fibrous tissue. The cuneiform bone is united by fibrous tissue to the ossified internal lateral ligament. (b) The semilunar bone has become completely fused, partly with the ossified articular disc and partly with the lower end of the radius. The line of fusion is partly fibrous, but there is for the most part complete bony union. The proximal row of the carpus consists, in consequence, of two bones only, the scaphoid and the cuneiform. (5) As a result of these changes the radio-carpal joint has disappeared, being represented only by a small joint between the radius and the scaphoid bone. (6) The opposing surfaces of the scaphoid and semilunar are united by fibrous tissue. There is, however, a joint between the semilunar and cuneiform bones which does not communicate with the transverse carpal articulation. (7) The synovial membrane of the transverse carpal joint extends normally between the bones of the proximal and distal rows, but has no prolongations between the bones of the distal row, all four of which are firmly united by fibrous tissue.

Right Femur.—This is a very big bone, and a detailed account of its measurements will be given later. The upper end appears bull-necked owing to the



FIG. 336.
Right femur.

enlargements of the head and neck. The trochanters are big, and there is a well-developed third trochanter. It will be seen from *Fig. 336* that the lower end of the diaphysis is of abnormal shape, the normal curves

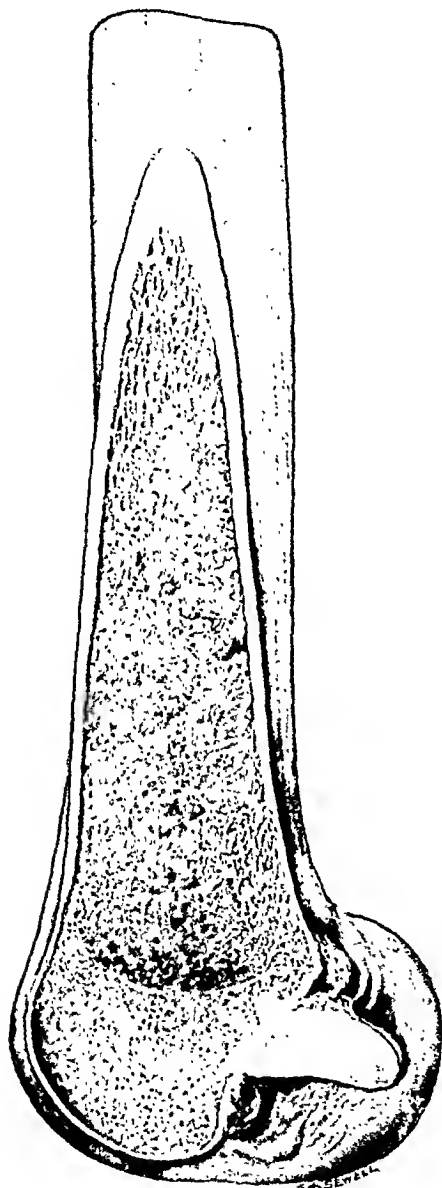


FIG. 337.—Left femur, showing bone developed in the capsule of the knee-joint.

by which the shaft suddenly splays out into the condyles being replaced by a general conical enlargement extending some way up the shaft. This enlargement is smooth, and its extent may be realized from the fact that the transverse popliteal measurement (p. 422) is only 1 mm. less than that possessed by the giant O'Brien, whose skeleton is in the Museum of the Royal College of Surgeons of England. There is a small nodular exostosis immediately above the external condyle, and the articular cartilage of the lower end shows a considerable degree of destruction, which was evidently a result of the ankylosis of the joint. Section of the bone shows: (1) An extensive atrophy of the bone-marrow, the whole of the medullary cavity being filled with fatty marrow except at the extreme lower end. (2) On the posterior surface, in line with the *linca aspera*, an area of new bone has developed, invading the medullary cavity, and reaching from just below the neck to the junction of the middle and lower thirds of the bone. It is composed of cancellous bone, and over it the normal compact bone of the femur appears to be somewhat thickened. Traversing this cancellous bone is a shaft of compact bone formed in the line of the nutrient artery. (3) In the lower half of the bone three transverse bony septa have developed, dividing the medullary cavity into a series of cylindrical compartments.

Left Femur.—Apart from its general size and shape, the interest of this bone lies in its exostoses, one of which arises from the upper margin of the

external condyle, one is developed in the muscular attachments to the adductor tubercle, and the third (*Fig. 337*) arises from the intercondylar fossa, and represents an ossification of the capsule of the knee-joint. It measures 2.3 cm. in length, and is of conical shape with a knobbed extremity.

Right Knee-joint.—This is illustrated in *Fig. 338*. In the infrapatellar tendon are many separate and irregular pieces of bone, while the patella is continuous with a large mass of cancellous bone developed in the quadriceps

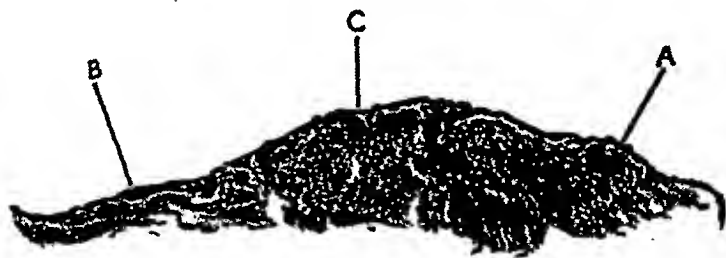


FIG. 338.—Right knee-joint. A, Bone in quadriceps muscle; B, Infrapatellar ligament; C, Patella.

muscle. The under surface of this mass is covered by cartilage for a distance of 1.5 cm. above the patella, and is partially subdivided by thick strands of fibrous tissue. During the dissection of the joint numerous pieces of bone unattached to the skeleton were removed. They varied in size from a pea to a mass weighing 26 gm.

Right Tibia and Fibula.—The tendons of the gastrocnemius and peroneal groups were rigid and gritty, owing to the deposition of calcareous material. At the upper extremity of the tibia are a large number of bony masses connected with the epiphysis, varying in size up to the large mass attached to the medial condyle (*Fig. 339, A*). The bigger masses possess a firm bony union with the tibia, while the smaller ones are united by fibrous tissue only, and are freely movable. A few of the loose masses consist entirely of cartilage. The history of their development leaves no doubt that these deposits of bone are not true exostoses, but areas of ossification in the ligaments and tendons attached to the head of the tibia, to which they have later acquired a bony union. Nine centimetres above the ankle-joint is a nodule formed by ossification in the interosseous membrane. The lower extremity possesses a well-marked deformity which is exactly repeated in the bones of the other leg. A bony outgrowth has developed in the anterior inferior tibiofibular ligament and the neighbouring part of the capsule of the ankle-joint, projecting downwards in front of the lateral malleolus (*Fig. 339, B*). It is of roughly circular shape, measuring 3.1 cm. and 2.7 cm. in its two diameters.

Left Tibia and Fibula.—The anterior half of the head of the tibia is continuous with an enormous bony



FIG. 339.—Right tibia and fibula. A, Bony masses at upper extremity of tibia; B, Bony out-growth at lower extremity.

mass (*Fig. 340, A*) developed in the infrapatellar tendon and the capsule of the knee-joint. It is very nodular, and has been evidently formed by the fusion of bones arising from numerous separate foci. The patella is firmly united to its upper extremity. Posteriorly it is almost entirely covered by articular cartilage. The union of this mass—which measures 23.5 cm. in circumference—with the tibia was still developing up to the time of death. Towards the lower end of the bone there is a small area of ossification in the interosseous membrane. The lower end of the left tibia closely resembles that of the right bone in the circular area of ossification in the capsule of the ankle-joint (*Fig. 340, C*), which is of somewhat larger size, measuring 4.7 cm. by 3 cm. in its two diameters. These two new masses are of disc-like shape with incurved edges.



FIG. 340.—Left tibia. *A*, Bone developed in capsule of knee-joint; *B*, Bone developed in peroneus longus muscle; *C*, Bone developed in ligaments of ankle-joint.

group, from the sheaths of which it presumably arose. It had no bony attachments, and was shelled out of the body by the fingers alone. It is composed of cancellous bone and weighs 27 gm.

Skull.—The vault of the skull was distinctly harder than usual to saw through. No abnormalities were found within the skull, except that on the right posterior clinoid process were two small exostoses of nodular shape rising to a height of 3 mm. above the level of the left posterior clinoid process. The sella turcica was normal in size and shape. From the external occipital protuberance a nodular process of bone extended into the ligamentum nuchæ, and there was a great deal of irregular ossification of the ligaments attached to the

A nodular mass of bone is attached to the fibula on its outer surface at the junction of the upper and middle thirds, projecting downwards for a distance of 1.7 cm. Section shows that it is not a true exostosis but has been developed in the muscular attachments of this region. A skiagram of the bone after removal of the soft parts shows a deposit of new bone lining the medullary cavity of the same type as that found in the femur. Two long conical pieces of bone were found lying free in the substance of the peroneus longus muscle close to its origin from the fibula (*Fig. 340, B*).

Right Foot.—The bone illustrated in *Fig. 341* lay in front of the right ankle-joint, posterior to the tendons of the long extensor



FIG. 341.—New bone in front of right ankle-joint.

base of the skull. *Fig. 342* shows these features, as indicated by the arrows, and in addition it can be seen that the frontal sinuses were well developed, and that the mental protuberance of the lower jaw was more prominent.



FIG. 342.—Skigram showing the changes in the skull.

than usual. The shadows seen in the cranial cavity were due to areas of ossification in the arachnoid membrane.

Vertebral Column.—This showed: (1) Bony outgrowths of nodular shape extending from the spines of the upper three cervical vertebrae into the ligamentum nuchae; (2) A diffuse process of ossification in the anterior longitudinal ligament over the right half of the bodies of the lower six dorsal vertebrae, causing a general enlargement of the right half of this part of the vertebral column compared with the left half.

Ribs.—The costal cartilages were not ossified. The only abnormality was an exostosis arising from the inferior surface of the neck of the third right rib close to the tubercle. It is of irregular ovoid shape, measuring 1.4 cm. in depth, and is seen in section in *Fig. 343*. It consists of a core of cancellous bone covered by a thin layer of compact bone, both of which are continuous with the corresponding parts of the rib, which is separated from the exostosis by a distinct notch. It has the typical structure of a true cancellous osteoma.

Pelvis.—There was a smooth diffuse enlargement of the crest of the ilium on the right side, due to ossification in the attachment of Poupert's ligament.



FIG. 343.
Section through exostosis
of rib.

Meninges.—The dura mater was normal, but there was extensive ossification of the arachnoid membrane. This reached its maximum in a smooth bone attached to the lateral and inferior surfaces of the right frontal lobe at its tip. This bone (the inner surface of which is seen in *Fig. 344*) weighs 4 grm., measures 3.2 cm. by 2.7 cm., and is about 1 mm. thick. The



FIG. 344.—Plate of bone developed in cerebral arachnoid.

bone has the general form of a thin bent plate, smooth on its outer side, irregular on its inner side, where there are three ridges which lay during life in the cerebral sulci. Elsewhere the cerebrum is covered by scattered deposits of bony plaques of similar nature, though of less extent. They were not found below the tentorium, and were all developed in the arachnoid membrane. In all, fourteen areas of ossification could be distinguished, covering a total area of sixteen square centimetres. On the whole they show a tendency to arise over the sulci. They gave rise to no symptoms during life. Microscopically the bone is contained within the arachnoid. This membrane shows no thickening in the immediate vicinity of the bone

formation, and there is no round-cell infiltration or other evidence of inflammation. The bone shows typical osteoid structure, with lacunæ and distinct lamination, but true Haversian systems cannot be detected. There is no cartilage present, and no tumour cells could be traced.

Endocrine Organs.—

The Suprarenals were enlarged (right 7.9 grm., left 6.1 grm.), but no abnormalities were found.

The Testes were normal to the naked eye, but unfortunately were not examined microscopically.

The Thyroid was diffusely enlarged, weighing 75 grm. The cut surface showed more colloid than usual, but was homogeneous except for two yellowish waxy nodules in the substance of the right lobe. A mass of similar appearance lay in the position of the left upper parathyroid gland, separated from the thyroid by a well-defined capsule. It was of ovoid shape, 2.5 cm. in length by 1.4 cm. in breadth, and weighed 2 grm. Apart from this, no bodies corresponding with the normal



FIG. 345.—Section of the thyroid, showing a small adenoma.

parathyroids were found. Histologically, the thyroid consists of vesicles of very irregular size, many of which are much larger than is usually met with, and from all these occur all gradations of size down to the smallest. The vesicles are all filled with colloid. In several places small adenomatous

collections of non-vesicular thyroid tissue of rather embryonic type are met with. One of them is shown in *Fig. 345*. The tumour found in the position of the left upper parathyroid is contained within a capsule of fibrous tissue and compressed thyroid tissue. The cells within the capsule are all of spheroidal type, and might serve either for parathyroid or for foetal thyroid cells. In part they are arranged in columns and in part in vesicles, with intermediate stages between the two. The vesicles contain colloid, staining like that of the thyroid. There are no eosinophil cells. A section of the tumour is seen in *Fig. 346*. Owing to its position, and to the fact that nothing corresponding to the normal parathyroids was found, the tumour gave rise to much difficulty in interpretation. In view of the type of the cells, and of the presence of similar adenomatous formations elsewhere in the thyroid, it is to be regarded as of thyroid, and not parathyroid, origin.

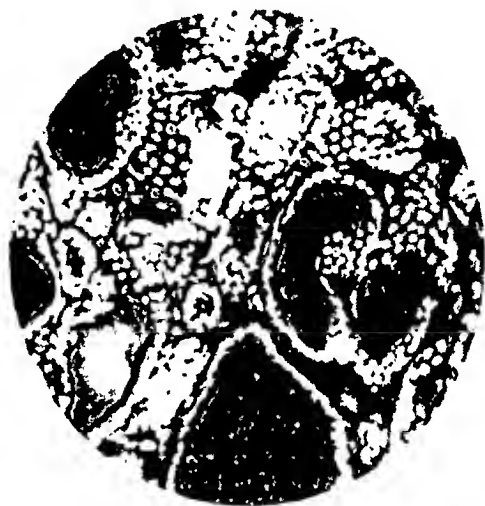


Fig. 346.—Section of the tumour found in the position of the left parathyroid gland.

The Pituitary (weight 0.55 grm.) was normal in size and shape. Professor Swale Vincent kindly investigated its histological structure, and I am much indebted

to him for the following report: "There is an increase in the eosinophil cells of the pars anterior. It is probable also that the pars nervosa is hypertrophied. It is also pretty certain that the colloid vesicles in the pars intermedia are increased both in size and number, though caution is necessary on the latter point owing to the extent of the normal variations."

The Bone-marrow showed a marked aplasia everywhere, the red marrow of the long bones being extensively replaced by an unhealthy-looking fatty marrow. Sections from the humerus and the right femur were examined by Professor O. C. Gruner, and I am greatly indebted to him for the following report: "It is clear that the marrow is specifically diseased, the changes taking the form of an extreme aplasia. The cells are dominantly of the lymphoid type, the formation of both myelocytes and of red cells being inhibited. The presence of numerous plasma cells suggests the existence of a chronic inflammatory process, which perhaps preceded the atrophy. In the case of the marrow from the humerus, some polychromatophil hæmoblasts and a few megakaryocytes were noticed. Degenerative changes were present in many of the hæmopoietic cells, and a number of chains of streptococci were present amongst these cells."

Structure of the Bones.—In handling and sawing the bones of this case it was noticed that they were both harder and more brittle than normal. These findings have been confirmed by all who have had to saw them. Pieces.

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It will be seen that the expectation of a high calcium content was not realized, and the explanation of the hardness and brittleness is to be sought in the histological structure. *Fig. 347*, which is a transverse section from the femur, shows the most significant change. There is a marked process of sclerosis, the bone itself being very dense, and there is extensive deposition of new bone inside the Haversian canals. In some the deposit is semilunar in shape, in others it has become circular, and in others it appears to have almost entirely filled up the canal, the process apparently proceeding to a sufficient degree to cause obliteration and thrombosis of the vessels.

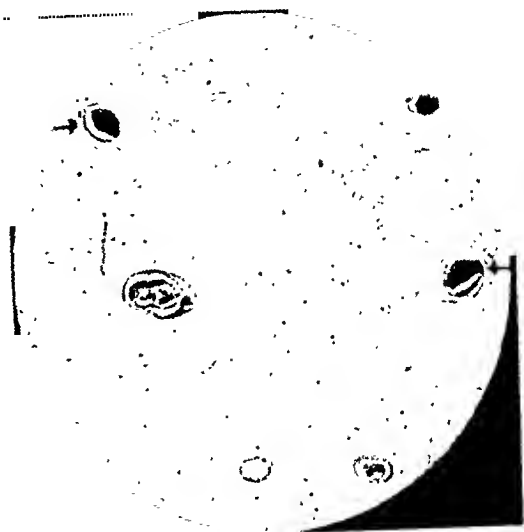


FIG. 347.—Transverse section from left femur. ($\times 45$.)

DISCUSSION.

Amongst the varied phenomena of which this case appears to consist, processes can be recognized which bring it into relation with certain general diseases of bone. These will now be discussed.

Ossification in Ligaments, Tendons, and Muscles.—This process accounts for some of the more striking manifestations of the disease, and was the first to attract attention clinically. It is especially evident in the region of the knees. In the muscles, deposition of bone has occurred in the rectus femoris on both sides, and in the peroneal group on the left side. In the tendons, the patellar tendon on both sides exhibits enormous deposits of bone, and in the arm a ridge of bone has been deposited in the attachments to the lateral ridge of the humerus. The peroneal tendons were undergoing calcification. The bone found in front of the right ankle-joint may also be added here, as it was probably developed in connection with the tendon-sheaths. In the ligaments there are the symmetrical deposits at the ankle-joints, the extensive deposits in the capsules of both knee-joints, including the outgrowth of compact bone from the left femur, the compact bone developed in the interosseous membrane of the forearm, the replacement by bone of part of the normal ligamentous structure of the wrist-joint, and finally the deposits in some of the ligaments of the vertebral column. The ligaments seem to have been the most susceptible of the three tissues, and the process was in activity up to the time of death.

This aspect of the case suggests a resemblance to progressive myositis ossificans, a rare disease of congenital origin in which bone is laid down in the fibrous tissues of muscles, tendons, ligaments, and aponeuroses. Although this often begins in the first year of life, in about 20 per cent of the

cases it has not appeared until the age of 10, when the symptoms began in this patient. It usually shows itself first by the presence of nodules in the muscles, which subsequently undergo fibrosis, and then ossification. In this connection, the earliest observation we have of the present case—that at the age of 11 the patellar tendon was studded with what were considered to be multiple fibromata—has some significance. In about one-third of the cases the skeleton is affected by a curious deformity, a condition of microdactyly which mainly affects the thumbs and big toes. This feature was not present here. In over 50 per cent of the cases the fusion of the ectopic masses of bone with the skeleton leads to the production of apparent exostoses, and this was a prominent feature in the present case. In some of the earlier case-reports it led to confusion with the disease known as multiple exostoses, but I have not succeeded in finding an instance of the association of progressive myositis ossificans with true cancellous exostoses such as are found in diaphysial aclasis. Had the features collected together under this heading occurred in an otherwise normal man, there can be little doubt that it would have been correct to classify them under the heading of progressive myositis ossificans.

The Changes at the Knee-joint.—The changes found at the knee-joints in this case bear a close resemblance to those found in a series of cases which have recently attracted attention in France. The condition was first described by Dujarier² in 1922, who reported three cases under the title of “Ostéomes juxta-tibiaux du Ligament rotulien”, since when other instances have been recorded. Areas of ossification are found in the lower part of the infrapatellar tendon, quite separate from the anterior surface of the tibia, which tends to be of irregular form. In Mouchet and Chakir Bey's case³ the process had gone a stage further, with bony union between the tibia and the new bone, as was found in the present case. The skiagram in their case brings out a further resemblance, in that stress is laid on the irregular ‘staircase’ appearance of the tibia behind the exostosis, from the condylar surface to the tuberosity. Maucclair⁴ states that the cases vary in severity from a simple osteoma in the tendon to its complete ossification, and at a discussion he showed skiagrams indicating complete ossification of the whole capsule of the knee-joint. The relation of this condition to progressive myositis ossificans does not appear to have been raised, but there can be no doubt that these observations are in general accord with the state of the knee-joints in the present case.

Ossification of the Cerebral Arachnoid.—The areas of ossification found in this region differ in no respect from the other instances that have been reported of this condition. It is not usual for the deposit of bone in the meninges to be accompanied by new bone formation in other parts of the body, though it occurs in some of the cases of the ‘marble disease’ (Albers-Schönberg) of bone, and ossification of the spinal arachnoid was reported by Hadden and Ballance⁵ in one case of acromegaly. A striking feature of the case-reports is the frequency with which the dura mater, particularly the falx and the sides of the longitudinal sinus, is affected, and its omission in this case, where new bone formation was so extensive, is remarkable. Ossification of the arachnoid and pia mater usually occurs in small plates,

with a smooth outer surface and a spiny inner surface, and tends to affect particularly the hemispheres. Pieces of bone as big as that found over the frontal lobe (*see Fig. 344*) are not common, though McKendree and Imboden⁶ state that the condition may be of such extent as to envelop the brain in a bony sheath moulded to the convolutions. The deposits do not give rise to symptoms, unless they develop into a large osteoma either pressing on or entering into the brain, as in a case reported by Meschede.⁷

It is curious that there is no record of the meninges being affected in this manner in cases of progressive myositis ossificans. The fact that the condition is generally discovered accidentally at the post-mortem, and that satisfactory post-mortem records of myositis ossificans are remarkable for their paucity, may be responsible for this. In the present case it is evident that the arachnoid shared in the same general tendency to bone formation that was evinced by the muscles and ligaments.

Exostoses.—It has already been mentioned that most of the apparent exostoses found in this case were really ectopic formations of bone which later acquired a secondary union with the skeleton. After these have been excluded, however, there still remain a few which have arisen within the skeleton and possess the structure of a cancellous osteoma. The exostoses on the right posterior clinoid process, on the right radius, and on the third rib, belong to this category. Their structure is clearly seen in the illustration of the rib (*see Fig. 343*), which brings out the characteristic feature that the cancellous core and the compact bony covering of the exostosis are both continuous with the corresponding parts of the parent bone. The presence of these exostoses at once brings the case into relation with a further disease of bone—diaphysial aclasis (multiple exostoses), and there are, in addition, further features which indicate that the relationship is a close one. These are the presence of malignant disease, and the changes found in the growth and shape of the long bones.

The Sarcoma of the Humerus.—The enormous growth in the right shoulder proved to be an osteosarcoma arising from the upper end of the humerus. Unfortunately there is no information as to whether there was a pre-existing exostosis at this place. Malignant disease occurs occasionally as a complication of other multiple diseases of bone, but it is particularly well known in diaphysial aclasis. Lenormant and Lecène⁸ have collected at least five cases of true secondary malignancy, the growths being chondrosarcomas which in three instances formed generalized deposits in the viscera. Many examples will be found in the recent monograph on this disease by Stocks and Barrington.⁹ It is difficult to estimate exactly the frequency with which this complication occurs, but both Ehrenfreid¹⁰ and Stimson¹¹ consider that about 5 per cent of the cases are affected. In progressive myositis ossificans, on the other hand, malignant changes are unknown.

CHANGES IN THE GROWTH AND SHAPE OF THE LONG BONES.

The long bones of this case are strikingly large, and their appearance at once suggests a condition of acromegaly or gigantism. This aspect of the case will be dealt with later, but in the meantime attention is drawn to

the tables of measurements of the right femur and tibia, when the great increase in absolute size over the normal mean measurements for these bones will be seen at once.

Table II.—MEASUREMENTS OF THE RIGHT FEMUR COMPARED WITH THE MEAN MEASUREMENTS OF THIS BONE AS GIVEN BY PROFESSOR KARL PEARSON.¹²

NATURE OF MEASUREMENT	MEASUREMENTS OF CASE	MEAN MEASUREMENTS
Maximum length	533.5 mm.	446.77 mm. \pm 1.02
Oblique length	531.5 „	444.35 „ \pm 1.03
Trochanteric length	490.0 „	426.06 „ \pm 1.02
Maximum trochanteric length ..	501.5 „	435.09 „ \pm 1.04
Oblique trochanteric length	489.0 „	423.55 „ \pm 1.06
Maximum vertical diameter of head*	58.2 „	47.06 „ \pm 0.14
Maximum horizontal diameter of head*	53.7 „	46.60 „ \pm 0.14
Minimum anteroposterior diameter (infratrochanteric)	38.4 „	26.83 „ \pm 0.08
Minimum transverse diameter (infratrochanteric)	36.0 „	31.68 „ \pm 0.13
Maximum anteroposterior diameter (shaft)	36.5 „	29.53 „ \pm 0.10
Maximum transverse diameter (shaft)	31.6 „	28.02 „ \pm 0.10
Bicondylar width	96.0 „	80.15 „ \pm 0.22
Minimum horizontal diameter of neck	33.4 „	25.74 „ \pm 0.09
Minimum vertical diameter of neck ..	43.3 „	33.84 „ \pm 0.12
Popliteal width (4 cm. above the upper margin of the articular surface of the external condyle)	56.2 „	39.83 „ \pm 0.17

* 2 mm. have been subtracted to allow for the articular cartilage.

It is evident from the tables given that the bones are not mere uniform enlargements of the normal shape, but that some parts are relatively more enlarged than others. This feature is of great importance, and is brought out in *Tables III* and *IV*. The mean measurements and indices of the femur are taken from Professor Karl Pearson's monograph on the English femur,¹² and I am indebted to him for permission to make use of his tables. Unfortunately there are no corresponding figures for the English tibia. Martin¹³ gives measurements of 2000 white American tibiae, and Wagner¹, in a recent monograph, measurements of 1100 Scandinavian tibiae. It was possible to collect only 31 corresponding English bones, but these were measured, and form the basis of comparison with the tibia of the case. After

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Table III.—TABLE OF INDICES OF RIGHT FEMUR COMPARED WITH MEAN INDICES AS GIVEN BY PROFESSOR KARL PEARSON AND INDICES OF AN ACROMEGALIC FEMUR.

VARIETIES OF INDEX	PRESENT CASE	MEAN	ACROMEGALY
1. Primary capital index $100 \times \frac{\text{horizontal diameter of head}}{\text{vertical diameter}}$	95.7	98.81 \pm 0.092	97.06
2. Secondary capital index $100 \times \frac{\text{vertical diameter of head}}{\text{maximum length}}$	10.909	10.51	11.59
3. Index of robusticity of head* .. $100 \times \frac{\text{sum of head diameters}}{\text{maximum length}}$	20.97	20.92	22.84
4. Third capital index $100 \times \frac{\text{vertical diameter of head}}{\text{bicondylar width}}$	60.6	58.81 \pm 0.127	62.19
5. Index of neck $100 \times \frac{\text{horizontal diameter of neck}}{\text{vertical diameter}}$	77.1	76.41 \pm 0.267	87.5
6. Index of robusticity of neck* .. $100 \times \frac{\text{sum of neck diameters}}{\text{maximum length}}$	14.35	13.3	15.34
7. Platymerie index $100 \times \frac{\text{platymerie anteroposterior}}{\text{platymerie transverse}}$	106.6	85.326 \pm 0.385	101.4
8. Pilastric index $100 \times \frac{\text{pilastric anteroposterior}}{\text{pilastric transverse}}$	115.5	105.80 \pm 0.418	119.3
9. Index of robusticity of femur .. $100 \times \frac{\text{sum of pilastric diameters}}{\text{oblique length}}$	12.81	12.96 \pm 0.037	15.56
10. Upper index of gracility $100 \times \frac{\text{pilastric transverse}}{\text{platymerie transverse}}$	87.7	88.8 \pm 0.243	86.83
11. Lower index of gracility $100 \times \frac{\text{pilastric transverse}}{\text{popliteal width}}$	56.23	70.74 \pm 0.290	81.5
12. Popliteal index* $100 \times \frac{\text{popliteal width}}{\text{maximum length}}$	10.53	8.91	8.65
13. Bicondylar width index $100 \times \frac{\text{bicondylar width}}{\text{maximum length}}$	17.99	17.94	18.6

* These indices are not given in Professor Karl Pearson's tables, but have been worked out from his mean measurements.

Continued on next page

Table III—continued.

VARIETIES OF INDEX	PRESENT CASE	MEAN	ACROMEGALY
14. Popliteal bicondylar index $100 \times \frac{\text{popliteal width}}{\text{bicondylar width}}$	58.54	49.88 ± 0.2	46.4
15. Direct oblique length index $100 \times \frac{\text{oblique length}}{\text{maximum length}}$	99.6	99.32 ± 0.020	
16. Direct oblique trochanteric index $100 \times \frac{\text{trochanteric oblique}}{\text{maximum trochanteric}}$	99.7	99.5 ± 0.023	
17. Primary capito-collal longitudinal index $100 \times \frac{\text{capito-collal axis}}{\text{maximum length}}$	14.25	17.22	

Table IV.—TABLE OF MEASUREMENTS AND INDICES OF THE TIBIA COMPARED WITH THE MEAN MEASUREMENTS OF 31 NORMAL TIBIÆ AND AN ACROMEGALIC TIBIA.

NATURE OF MEASUREMENT	MEASUREMENTS OF CASE	AVERAGE MEASUREMENTS	ACROMEGALIC MEASUREMENTS
Length	437.6 mm.	364.9 mm. (S.D. = 29.5 ± 2.53)	371 mm.
Width across the condyles ..	91.95 mm.	70.9 mm. (S.D. = 6.22 ± 0.532)	85.5 mm.
Thickness through shaft in region of tuberosity	65 mm.	43.7 mm. (S.D. = 4.9 ± 0.42)	47.9 mm.
Antero-posterior diameter of the lower extremity	50.4 mm.	38.2 mm. (S.D. = 3.29 ± 0.275)	34 mm.
1. $100 \times \frac{\text{condylar width}}{\text{length}}$	21.01 mm.	19.43 mm.	23.04 mm.
2. $100 \times \frac{\text{thickness at tuberosity}}{\text{length}}$	14.85 mm.	11.97 mm.	12.9 mm.
3. $100 \times \frac{\text{A P dia. of distal extremity}}{\text{length}}$	11.51 mm.	10.49 mm.	9.17 mm.
Sum of indices 1 and 2	35.86 mm.	31.4 mm.	35.94 mm.

S.D.=Standard deviation.

this had been done, it was found that Mr. Quarry Wood¹⁵ had done the same for 40 Scottish bones. His figures agree closely with mine. The acromegalic measurements given in these tables were obtained from a skeleton in the museum of the Royal College of Surgeons of England.

The Femur.—*Table III* gives the following information :—

1. The head has a greater vertical diameter relatively to the horizontal diameter than normal, and this feature is also shown by the acromegalic bone. Relatively to the length of the femur, the vertical diameter of the head is slightly in excess of normal, and the relative robusticity of the head (*Index 3*) is also slightly greater than normal, but in neither respect is the divergence from the normal so great as that shown by the acromegalic bone. Comparing the size of the upper epiphysis with the lower (*Index 4*), the head shows a greater relative increase than the bicondylar region, and here also the patient's bone occupies an intermediate position between the normal and the acromegalic bones.

2. A similar feature is shown by the measurements of the neck. In the relative increase of the horizontal diameter, and in the robusticity of the neck compared with the length of the bone, the present case is abnormal, but not so abnormal as is the acromegalic. This is particularly so in the case of the horizontal diameter.

3. As seen in *Fig. 336* the upper end of the femur appears bull-necked. *Index 17* reveals a shortening of the capito-collar axis, with the result that the head and neck are closely set on the shaft.

4. In the infratrochanteric region the present case is markedly abnormal, the anteroposterior diameter being 106 per cent of the transverse, instead of the normal 85 per cent. The acromegalic bone exhibits the same feature to a less extent (101 per cent).

5. In the middle of the shaft (*Index 8*) the same relative increase in the anteroposterior diameter is found, but here the figure for the case, though very abnormal, does not reach the same degree as the acromegalic. *Index 9* shows that in spite of the great size of the shaft anteroposteriorly, the total robusticity of the shaft is within normal limits, in this respect contrasting with the acromegalic.

6. In the popliteal region the bone exhibits a marked divergence from the normal which is not found in the acromegalic bone. The index of popliteal width (*Index 12*), measured 4 cm. above the external condyle, is 10.5 per cent, as against a normal 8.9 per cent and the acromegalic 8.6 per cent. This indicates a marked relative enlargement of the lower end of the diaphysis. This abnormality is also brought out in *Indices 11* and *14*. The enlargement of the lower end of the diaphysis is clearly shown in *Fig. 336*.

7. The lower epiphysis (*Index 13*) shows practically no relative increase in size, contrasting in this respect with the acromegalic, in which there is a slight increase.

It is thus apparent that the femur shows a series of abnormalities in addition to its great size. In the relative size of the head and neck and in the increase of the anteroposterior diameters of the platymeric and pilastric regions of the shaft it tends to follow the changes found in the acromegalic bone, though (with the exception of *Index 7*) to a less extent. The enlargement of the popliteal end of the diaphysis is in sharp contrast with the acromegalic bone.

The Tibia.—The following information may be gained from *Table IV* :—

1. There is a striking increase in the absolute size of all the measurements.

2. The size of the upper epiphysis, as measured by its transverse width, shows a relative increase over the normal, but—as with so many of the femoral measurements—it occupies an intermediate position between the normal and the acromegalic measurements.

3. The anteroposterior diameter of the shaft in the region of the tuberosity is greater than the normal and the acromegalic bones. (The tuberosity itself is developed from the upper epiphysis, so that this measurement is really partly epiphysial and partly diaphysial. In the patient's bone, however, the tuberosity does not project unduly, while the shaft is obviously big (*see Fig. 310*), and the increase in this index is undoubtedly due to enlargement of the upper end of the diaphysis.)

4. If *Indices 1* and *2* are added together, it is interesting to find that while both the patient's and the acromegalic bones show an increase in the size of the upper end of the bone of almost identical degree (35.8 per cent and 35.9 per cent, as against a normal 31.4 per cent), in the case of the acromegalic the increase is mainly due to the epiphysis, and in the present case to the upper end of the diaphysis.

5. It was not possible to make a transverse measurement of the lower end of the tibia, as it was still articulated with the fibula. Anteroposteriorly, the lower epiphysis is somewhat bigger than normal, while curiously enough in the acromegalic it is smaller.

The changes in the size of the various regions of the tibia may be summed up as a relative enlargement of both epiphyses and of the upper end of the diaphysis. In the upper epiphysis the abnormality is not so advanced as in the acromegalic, while in the upper diaphysis it is more advanced.

Relation of the Present Case to Diaphysial Aclasis.—The presence of true cancellous osteomata at once brings this case, as has been shown above, into relation with diaphysial aclasis. The presence of an osteosarcoma of the upper end of the humerus deepens that relation. The measurements discussed in the previous section provide yet another point of contact. Although the shape of the long bones appears in many respects to approach an acromegalic condition, changes are seen at the ends of the diaphyses which greatly exceed anything found in the acromegalic bones, and I am of the opinion that the great enlargement of the lower end of the femoral diaphysis and the upper end of the tibial diaphysis should be considered in connection with diaphysial aclasis. In this disease, as Sir Arthur Keith¹⁶ has pointed out, changes are found in the shafts of the long bones, at the ends of the diaphyses, which are due to a disturbance in the modelling function of the periosteal bone which rounds off the central core of cartilaginous bone into its final shape. The ends of the diaphyses tend in consequence to be big and clumsy. One cannot but associate with this process the enlargements of the diaphysial ends of the femur and tibia which have just been described.

Relation of the Case to Acromegaly.—There are certain features which indicate that in yet another of its aspects this case must be considered in relation to acromegaly: (1) The general character of the changes in the

growth and shape of the long bones is very suggestive. In addition to the increase in absolute size, the detailed measurements show a striking approach to the condition found in the acromegalic skeleton. Acromegaly is a disease of slow progress, and the intermediate character of the measurements of this case would be explicable by the early death of the patient. As the process was apparently at work before the closure of the epiphysial lines, there is a mixed element of gigantism and acromegaly. (2) There was no enlargement of the soft tissues, but the hands and feet were large in proportion to his size, and, when shown at the Royal Society of Medicine, several observers commented on his acromegalic appearance. (3) The skiagram of the skull shows well-developed frontal sinuses, and the protuberance of the chin is decidedly prominent. (4) Additional evidence is provided by the organs of internal secretion. Though the pituitary was of normal size, among the abnormalities described by Professor Swale Vincent was an increase in the eosinophil cells of the anterior lobe. The pituitary adenomata usually found in cases of acromegaly are composed of these cells, and where there is no adenoma a hyperplasia of the eosinophil cells is generally found. The common change in the thyroid is an increase in size, with the presence of a colloid goitre containing adenomatous nodules (Cushing and Davidoff¹⁷), such as was found in this case. Thus the changes in the bones suggest a mixture of acromegaly and gigantism, while the changes found in the organs of internal secretion are of the type associated with these diseases. One cannot but conclude that processes were at work in the case identical with those that are responsible for acromegaly and gigantism elsewhere.

The Aplasia of the Bone-marrow and the Presence of an Infective Element.

—The extensive atrophy of the red marrow was a surprise, as the two blood-counts had shown a slight degree of polycythæmia. Dr. Gruner suggests in his report that the bone-marrow was the seat of a chronic inflammatory process prior to the atrophy. It is probably this which links the aplasia to the rest of the case, as inflammatory changes had been present in the ulna for two years before death. The presence of sepsis is not remarkable, for in such a closely related condition as diaphysial aclasis sepsis is known to occur at times (Davis¹⁸). The case as a whole cannot be ascribed to an infective origin, as, apart from the bony lesions in the father, the disease had produced clinical evidence of its presence for seven years before the onset of inflammatory changes.

The Unilateral Nature of the Lesions.—The case appears to show a definite tendency for the right side to be affected more than the left. The condition began at the right knee, the left knee remaining unaffected for five years. The bony deposits in the arachnoid were bilateral, but the largest plaque developed over the right frontal lobe, and it is probable that the ossification of the arachnoid began here. The pelvis and ribs were affected only on the right side, and in the vertebral column the ossification of the anterior longitudinal ligament over the lower dorsal vertebræ was sharply limited to the right side. The left upper limb was normal, while the right showed changes from the clavicle to the carpus. It should be added that this feature does not apply to the size of the bones, for measurements

show that the leg bones were equally affected in this respect. The presence of such a unilateral tendency points to a developmental disorder as the fundamental basis of the condition.

COMMENTARY.

Many of the features shown by this case are due to the close similarity between certain of its aspects and the three disorders of bone formation that have already been discussed. Others may be attributed to a chronic infective element appearing towards the end. The changes at the clavicle and the wrist-joint appear to be quite anomalous, and it is suggested that the condition may represent a clinical entity which should be looked for again. Its relation to the above three diseases is of great interest, and there can be little doubt that these aspects of the case were produced by a combination of factors which, occurring singly as in the ordinary way, produce one or other of these diseases. The following hypothesis is put forward as a possible explanation.

The presence of the bony abnormalities in the father indicates that the disease had an hereditary basis—i.e., that it reflected in some way an abnormality present in the zygote. It is not a little curious that this abnormality, in working out its clinical expression, should have led, amongst other things, to the production of changes apparently typical of three disorders of bone, in each of which there is some reason for postulating an abnormality dating back to the original germ plasm.

1. *Progressive myositis ossificans* is undoubtedly of congenital origin. This view of its nature appears to be necessitated by the fact that in about a third of the cases it is accompanied by a defect of regular character in the formation of the skeleton (microdactyly). Burton-Fanning's case,¹⁹ in which both the muscular changes and the microdactyly were passed on from father to son, provides strong additional evidence. The experimental study of genetics, such as the work of Morgan,²⁰ would suggest that in the zygote there was an error in one of the chromosomes, possibly quite a localized one, affecting one or more genes, and such an original error may underlie all cases of this disease.

2. The evidence that *diaphysial aclasis* depends on a chromosomal abnormality is much stronger, for an hereditary factor can be recognized in about 75 per cent of the cases, traceable in a few instances through three or four generations. A chromosomal error would appear to be the only explanation of this disease consistent with the results derived from the study of genetics, though the means by which such an original error produces in later years the characteristic clinical picture—whether there is or not an intermediate mechanism in the organs of internal secretion—is uncertain.

3. The direct evidence that *acromegaly* and *gigantism* may also be expressions of an original chromosomal error is weaker, but the close relation between the two conditions, coupled with the fact that *gigantism* is a developmental disorder, is suggestive. Further, cases of hereditary and familial *acromegaly* have been reported, including one in which the father, sister, and brother of

the patient showed acromegalic changes (Fraenkel, quoted by Atkinson²¹). It would be consistent with our present knowledge of acromegaly to regard it as a congenital condition, represented in the zygote by an abnormality whose subsequent development produces the ordinary clinical appearances through alterations in the pituitary gland and other organs of internal secretion.

It is certainly remarkable that the tendency to abnormal development of bone inherited by the patient should have partly expressed itself by producing a series of changes such as are found in three diseases, each of which it is possible to regard as reflections of an original chromosomal error in the zygote, and all of which are alike in a tendency to uncontrolled formation of bone. It is unlikely that their presence here is a coincidental grouping of morbid events, and it is suggested that the case depends on a chromosomal alteration in the germ plasm, presumably a mutation affecting the part or parts that may be concerned in these three diseases, but of a more extensive nature, which in its subsequent development has in consequence produced certain anomalous features in addition to those just referred to. The suggestion is attractive in that it provides a plausible explanation for the present case as well as suggesting that the diseases in question may not be entirely unrelated phenomena. They may indeed all depend on an area of the germ plasm, such as a gene or series of genes in one chromosome, liable at times to go astray in one of these three ways. Until it is possible to provide for the human being a chromosomal map such as Morgan has provided for *Drosophila*, this must remain a speculation. It may, however, be pointed out that we already know of such spatial relationships in disease, the abnormalities for hæmophilia and night blindness being both situate in the X chromosome. Progressive myositis ossificans, diaphysial aclasis, and acromegaly may, in the light of this case, have some similar relation. It is possible that they may depend on genotypic mutations producing their effect by way of the organs of internal secretion. There would appear to be visible evidence of this in the case of acromegaly, where the changes in the pituitary and thyroid are prominent. Very little is known with regard to myositis ossificans and diaphysial aclasis in this respect, and it may be that as a genotypic abnormality would be reflected in the structure of every cell in the body, the alterations in these disorders may be chiefly potent in the fibrous tissues of muscles and periosteum.

While it is claimed that the present case represents an undescribed disease of bone, it is to be regarded, not as an isolated curiosity, but as a condition which may throw light on the nature and possible inter-relationship of these three diseases.

It is impossible to mention individually all who have helped me, but in addition to those already named in the text, my thanks are due to Dr. A. D. Millington, of Woodsetton, with whom I first saw the case; to Dr. G. E. Dyas, Radiologist to the Wolverhampton and Staffordshire Hospital, for the unstinted help given by his department, and to Dr. S. C. Dyke, Pathologist to the Hospital, who has very kindly provided me with the histological descriptions which have been incorporated in the text. At the Royal

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REFERENCES.

- ¹ SHELDON, J. H., *Proc. Roy. Soc. Med. (Clin. Sect.)*, 1925-6, xix, 7.
- ² DUJARIER, C., *Médecine*, 1922, iv, 28.
- ³ MOUCHET and CHAKIR BEY, *Bull. de la Soc. Anat.*, 1924, 6th s., xxi, 110.
- ⁴ WIART, MAUCLAIRE, and DUJARIER, *Bull. et Mém. Soc. de Chir.*, 1921, xlvii, 1167.
- ⁵ HADDEN, W. B., and BALLANCE C. A., *Clin. Soc. Trans.*, 1888, xxi, 201.
- ⁶ MCKENDREE, C. A., and IMBODEN, N. M., *Arch. of Neurol. and Psychiat.*, 1921, vi, 529.
- ⁷ MESCHKE, *Virchow's Arch.*, 1866, xxxv, 472.
- ⁸ LENORMANT and LECENE, *Rev. d'Orthop.*, 1906, vii, 203.
- ⁹ STOCKS and BARRINGTON, *Eugenics Laboratory Memoirs*, xxii, Vol. iii, *Hereditary Disorders of Bone Development*, Part I.
- ¹⁰ EHRENFREID, A., *Jour. Amer. Med. Assoc.*, 1915, lxiv, 1642.
- ¹¹ STINSON, P. M., *Arch. of Pediatrics*, 1917, xxxiv, 338.
- ¹² PEARSON, KARL, and BELL, JULIA, *Drapers' Company Research Memoirs*, Biometric Series x, Part I, "The Femur", 1919.
- ¹³ MARTIN, R., *Lehrbuch der Anthropologie*, Jena, 1914.
- ¹⁴ WAGNER, K., *Mittelalter Knochen aus Oslo*, Oslo, 1927.
- ¹⁵ WOOD, W. Q., *Jour. of Anat.*, 1920, liv, 232.
- ¹⁶ KEITH, SIR ARTHUR, *Jour. Anat. and Physiol.*, 1920, liv, 101.
- ¹⁷ CUSHING, H., and DAVIDOFF, *Reports of the Rockefeller Institute*, No. 22.
- ¹⁸ DAVIS, G. G., *Amer. Jour. Orthop. Surg.*, 1905-6, iii, 234.
- ¹⁹ BURTON-FANNING, F. W., *Lancet*, 1901, ii, 849.
- ²⁰ MORGAN, *The Physical Basis of Heredity*, Philadelphia, 1919.
- ²¹ ATKINSON, F. R. B., *Acromegaly*, Unpublished Monograph in the Library of the Royal Society of Medicine, London.

**A REPORT ON A SPECIMEN OF SPONDYLOLISTHESIS
FOUND IN THE SKELETON OF A BANTU NATIVE
OF SOUTH AFRICA :**

**WITH FURTHER SPECIMENS ILLUSTRATING AN ANOMALOUS MODE
OF DEVELOPMENT OF THE LOWER LUMBAR VERTEBRÆ.**

By L. R. SHORE,

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG, SOUTH AFRICA.

THE condition of spondylolisthesis is perhaps sufficiently uncommon to justify a short note. The collection of Bantu skeletons in the Anatomy Department of the University of the Witwatersrand has furnished not only a specimen of spondylolisthesis but also some others which illustrate a variation in the mode of development of the lower lumbar vertebræ which is, perhaps, not commonly recognized even by anatomists. In the specimen of spondylolisthesis to be described here, the abnormal mode of development referred to seems to be a causative factor in its production.

The following notes descriptive of these specimens are therefore presented in the hope that they may be found of interest to clinicians generally, and particularly to the orthopædist or to the obstetrician. It is to be regretted that prepared dried skeletons only form the material on which these notes are based, and that clinical notes are lacking, or limited only to a statement as to the cause of death.

Ziegler¹ describes spondylolisthesis in the following terms: "It is a deformity in which, by the action of the weight of the trunk, the body of the 5th lumbar vertebra and the portion of the spinal column above it slip forward over the base of the sacrum." It is also stated that the 5th lumbar vertebra comes to rest with its basal surface upon the ventral aspect of the sacrum, while its dorsal surface lies nearly on a level with the basal surface of the sacrum. Further, the vertebral arch with its spinous process does not in fact take part in the spondylolisthesis, the anterior half or body of the vertebra alone being displaced. As causative agents Ziegler suggests traumatic violence, fracture, inflammation, and anomalies of development in the laminae and interarticular parts of the arch.

Specimen I: Spondylolisthesis.—The specimen of the lumbar vertebræ and the sacrum was derived from the skeleton of a Bechuana woman, age 42 (No. 248).

The dried skeleton shows displacement of the body of the 5th lumbar vertebra downwards and forwards to a position in front of the sacral promontory. The dorsal arch, separated from the body, is slightly displaced from its position on the back of the sacrum to the superior surface, the position normally occupied by the body. The dorsal arch remains attached to the

dorsum of the sacrum by a mass of bone which is evidently of new formation and which considerably obscures the details of the arch. The displacement of the arch is estimated as 6 mm. in a forward direction.



FIG. 348.—Showing displacement forwards of 5th lumbar vertebra on the sacrum.

As *Figs. 348 and 351* show, the body of the 5th lumbar vertebra is supported in a position sloping downward and forward by a shelf of thin bone which is based on the upper anterior edge of the sacrum and which is also of new formation. The inferior surface of the vertebra now faces backwards and downwards. The slipped vertebral body is separable both from the sacrum and from the bony shelf that supports it. The upper surface of the sacrum is curved, with a superior convexity, as if it had been moulded in the passage of the slipped vertebral body over it. *Fig. 349* shows the front view, in which the body of the 5th lumbar obscures almost the whole of the first piece of the sacrum as it lies in its bony shelf. In *Fig. 350* the body of the vertebra has been lifted away in order to show the bony shelf which supported it. On the summit of the sacrum the dorsal arch can be distinguished.



FIG. 349.—Front view of *Fig. 348*.



FIG. 350.—Fifth lumbar vertebra removed to show the bony shelf on which it rested.

Examination of the specimen shows that the pedicles and the superior articular processes remain attached to the vertebral body, but that the inferior articular processes remain with the dorsal arch. Evidently, separation

of the dorsal arch has come about by division of the laminae between the superior and the inferior articular processes.

The positions of the outgrowths of new bone are worthy of note. Although, in general, exostoses of the vertebral column are very common in Bantu skeletons, in the present one they are limited to the positions already stated: (1) Between the dorsal arch of the 5th lumbar vertebra and the dorsal arch of the first piece of the sacrum; and (2) The anterior edge of the 5th lumbar vertebra. These exostoses are probably to be regarded as a reaction of the tissues to the abnormal stresses and strains that follow dislocation of the vertebral body.

There is only slight distortion of the bodies of the lumbar vertebrae except for the body of the 5th. This has become somewhat wedge-shaped, with the thick edge forward (Fig. 351), and the upper surface has become expanded—as it were, slightly 'mushroomed'.

The following phenomena are to be interpreted as evidence of abnormal strains:—

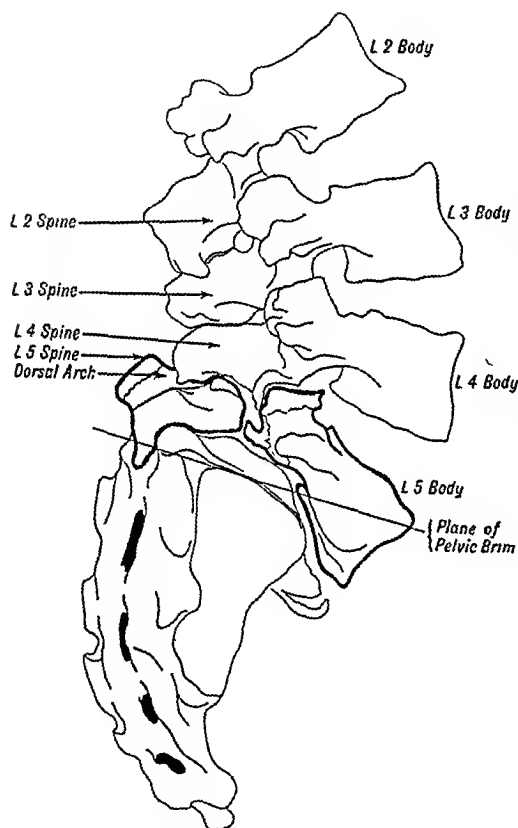


FIG. 351.—Diagram of reconstruction of the lower part of spinal column.



FIG. 352.—Reconstruction of the lower part of spinal column.

1. The inferior articular processes of the 4th lumbar vertebra are deformed. The articular surfaces are subdivided horizontally. The more posterior ones seem to be of new formation for as the 5th lumbar articular surface after dislocation had taken place.
2. Smooth areas, defined by circumferential osteophytes on the lower aspect of the 2nd lumbar spine and on the upper aspect of the 3rd and 4th spines. These seem to be areas of previous pressure. Their presence suggests that in life the lumbar spines had been locked at these places.

3. There is marked evidence of osteo-arthritis in the joints between the 2nd and 3rd and between the 3rd and 4th vertebræ. The joint between the 4th and 5th vertebræ shows the same condition.

4. There are also osteophytes on the ilia in the neighbourhood of the sacro-iliac joints.

Using the contact points described above as guides, an attempt has been made to reconstruct the lower part of the spinal column; the result is shown in a lateral dioptograph tracing in *Fig. 351*. A reconstruction of the pelvic skeleton was effected. A dioptograph tracing of the pelvic brim is shown in *Fig. 352*.

The following measurements of the pelvic brim were made, and an attempt was made to deduce the original value of the antero-posterior diameter before the dislocation of the 5th lumbar vertebra took place. Both these measurements are set out:—

Antero-posterior Diameter.—Front of 5th lumbar vertebra to symphysis pubis, 8.0 cm.; summit of sacrum to symphysis pubis, 11.4 cm.

Transverse Diameter.—11.5 cm.

Oblique Diameter.—11.5 cm.

It seems that dislocation of the body of the 5th lumbar vertebra brought about a reduction in the antero-posterior diameter of 3.4 cm., or $1\frac{1}{2}$ in. Unfortunately only the scantiest of clinical data referring to the individual are available. Delayed labour followed by rupture of the uterus and peritonitis are stated as the cause of death.

The next four specimens to be described are illustrative of the condition of congenital division of the laminae, which was a feature of this case of spondylolisthesis, and which, it is suggested, makes possible the separation and dislocation of the vertebral body from the dorsal arch.

Specimen II.—This specimen was obtained from the skeleton of a male Griqua, age 57 (No. 303).

It has been possible to prepare a reconstruction of the 5th lumbar vertebra and the sacrum in their natural positions with more than usual accuracy, by using as a guide the interlocking exostoses which arise from the lower border of the 5th lumbar vertebra and the upper edge of the sacrum.

Fig. 353 shows a dorsal view. Above, the superior articular processes of the 5th lumbar vertebra are easily identified. Immediately below each of these processes is a horizontal cleft on each side of the dorsal arch, passing through the lamina. The 5th lumbar spine in the mid-line can be distinguished from the dorsal ridge of the sacrum by its greater thickness and its expanded lower end, as well as by the space that separates these structures. This spine is supported by a plate of bone which is roughly quadrate in shape, and which is the separated dorsal arch of the 5th lumbar vertebra. The lateral edges of this arch are defined by the inferior articular processes which the arch bears posteriorly.

This specimen is similar to *Specimen I* in the separation of the dorsal arch, and may be regarded as illustrating a stage in the development of spondylolisthesis before dislocation has occurred.

Fig. 354 shows an anterior view, in which a fringe of small exostoses can be seen on the upper edge of the sacrum. In addition, there is a spur of bone on the right, and a much larger one on the left side, which has come into contact with a down-growing exostosis from the 5th lumbar vertebra.

Some exostoses arise from the upper edge of the 5th lumbar vertebra, and also from other members of the same column, but in no place are exostoses so prominent as at the lumbo-sacral junction. As the lumbo-sacral junction is a position which, as a general rule, is free from exostoses, their presence in this region is perhaps to be interpreted as evidence of some unusual strains, although the 5th lumbar is not actually dislocated.

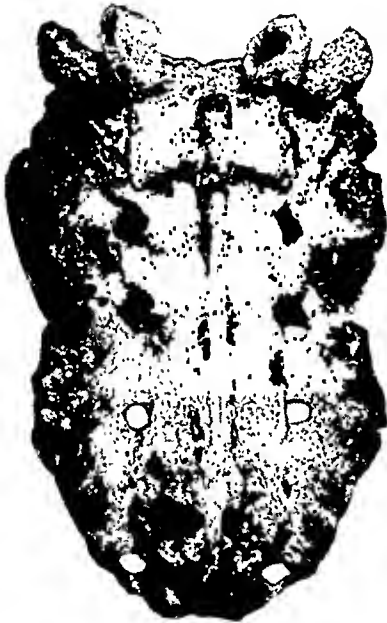


FIG. 353.—Dorsal view of *Specimen II* showing division of the laminae.

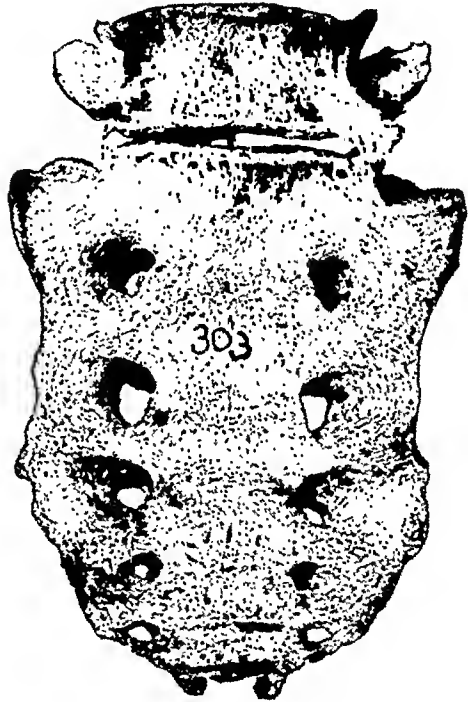


FIG. 354.—Anterior view of *Specimen II*.

Specimen III.—This specimen comprises the 4th and 5th lumbar vertebrae and the sacrum from the skeleton of a male Shangaan, age 60 (No. 307). These three bones were entirely separate, and for the purposes of photography a reconstruction of the intervertebral discs was made with the aid of plasticene. A slip of white paper was inserted in the spinal canal of the sacrum to show the superior hiatus.

Fig. 355 exhibits the dorsal view. The dorsal arch of the 4th lumbar vertebra shows a condition almost exactly like that described in *Specimen II*. The dorsal arch, bearing the inferior articular process and the spinous process, is separated from the rest of the 4th lumbar by a cleft in the laminae immediately behind the superior articular processes. The dorsal arch in the dried skeleton is an entirely separate bone.

Unfortunately, in the course of preparation of the skeleton, the piece of bone corresponding to the dorsal arch of the 5th lumbar has been lost and



FIG. 355.—To show the dorsal view of Specimen III.

The dorsal arch is completely separate from the rest of the vertebra. For the purposes of photography the spinal canal was filled with plasticine, and the dorsal arch restored to its natural position. The photograph has been taken with the spine directed downwards. From the spine the two laminae can be seen diverging. Following the laminae towards the body of the vertebra, a pair of prominences are met, which are the inferior articular processes and bear the articular surfaces on their lateral aspects, facing backwards and outwards. Immediately in front of the articular processes is a cleft in each lamina, which separates the prominence of the inferior articular process from a second prominence, which joins the pedicle and the transverse process and which bears the superior articular surface on its upper aspect.

has not been recovered even after careful search. Examination of Fig. 355 will, however, leave no doubt that an exactly similar condition to that found in the 4th lumbar must have been present in the 5th lumbar also. The transverse processes and superior articular processes of the 5th lumbar can be seen, and just medial and inferior to the last the truncated bases of the laminae. Below can be seen the superior sacral hiatus with the superior articular processes on either side of it. The missing bone must have comprised the inferior articular processes, the laminae, and the spine of the 5th lumbar vertebra. In this skeleton it is assumed that separation of the dorsal arch due to congenital division of the laminae took place in both the 4th and 5th lumbar vertebrae.

Specimen IV.—The skeleton that provided this specimen was that of a male Shangaan, age 36 (No. 280).

The only abnormal member of the vertebral column was the 5th lumbar, of which the inferior view is shown in Fig. 356.



FIG. 356.—Specimen IV, showing the abnormal 5th lumbar vertebra, inferior view.

which bears the superior articular surface on its upper aspect.

The vertebra is not quite symmetrical; the lamina is somewhat wider on the left side, the left pedicle is somewhat the thicker, and the spine is slightly deflected to the left. The vertebral body is more bulky on the left side. There is no evidence of any dislocation or of any pathological change in the vertebral column.

Specimen V.—This specimen was obtained from the skeleton of a male Xosa, age 45 (No. 159).

Fig. 357 shows the inferior aspect of the 4th lumbar vertebra, which was the only abnormal member of the vertebral column. The right lamina only is divided, actually slightly nearer to the superior than to the inferior articular process. On the left lamina, in a position corresponding exactly to this division, is a rough line on the surface of the bone (not shown in *Fig. 357*) which is more easily traced on the anterior aspect. This vertebra displays an incomplete separation of the dorsal arch, interesting for comparison with the specimens already described.

Asymmetry is very marked in this vertebra. The left lamina is much thicker and somewhat longer than the right, with the result that the inferior articular process of the left side projects laterally beyond the superior articular process, and even beyond the mammillary process of the same side.

Fig. 357 shows that the left lamina is reinforced on its anterior surface by a bar of bone that extends from the pedicle to the middle of the dorsal arch. The left pedicle is stouter than the right, and the body is more bulky on the left side. The spine also is slightly deflected to the left. It seems obvious, from a study of the asymmetry, that the intervertebral stresses were transmitted mainly by way of the left arch and pedicle. The vertebral column was free from obvious pathological change.



Fig. 357.—*Specimen V*, showing division of the right lamina of the 4th lumbar vertebra.

DISCUSSION.

The question obviously arises whether the separation of the dorsal arch is the outcome of developmental variation or of fracture. In every specimen described the situation of the cleft is distal from the position of the neuro-central joint of the immature vertebra. A post-mortem fracture must inevitably exhibit sharp or irregularly spiked edges and an exposed marrow cavity. These conditions did not obtain, and the explanation of post-mortem fracture is inadmissible.

In opposition to the explanation of ante-mortem fracture the following arguments may be brought forward: (1) If a fracture, the condition must be supposed to be the result of direct violence transmitted through the spinous

process. In this event the unilateral condition found in *Specimen V* would be a highly improbable consequence. (2) The position of the line of division is proximal to where a fracture of the lamina would be expected as the result of direct violence. (3) Some signs of repair would be expected after fracture. There is no evidence of callus formation; the surfaces of the bone in every case show no thickening in the region of the cleft, but are smooth and even to its edges.

An alternative mode of development of the lower lumbar vertebrae is described by Professor Peter Thompson,² who contributes the section on osteology to Morris's *Treatise on Anatomy*: "The 5th lumbar exhibits in some cases a special mode of ossification in the arch. Instead of two centres there are four—one for each side of the root, transverse process and superior articular process; and another on each side for the lamina, inferior articular process and the lateral half of the spinous process." This phenomenon described by Professor Thompson explains exactly the condition found in the specimens described in this paper. The separated dorsal arch consists of the laminae, inferior articular processes, and the spine, and must be supposed to arise by a lack of fusion between the two ossific processes from which each half of the arch is sometimes developed.

This same condition is recorded by T. A. Willis,³ of the Western Reserve University, Cleveland, Ohio. This observer has made a study of the vertebral column of man in the very large collection of human skeletons in the Hamann Museum. The material surveyed by Willis included 748 spinal columns derived from American white and negro individuals. Thirty-one specimens were found in 748 columns—that is to say, 4.28 per cent of vertebral columns exhibit the condition of congenital division of the vertebral lamina. Unilateral division of the lamina (on the right side in every instance) was found in 8 vertebrae, and bilateral division in 23. Willis also describes spondylo-listhesis and the association of spina bifida with congenital division of the vertebral lamina.

In Willis's series congenital division of the lamina occurred in the 3rd lumbar vertebra in 2 columns and in the last pre-sacral in 29 columns. The last pre-sacral vertebra was the 24th, i.e., the 5th lumbar, in 26 of the 29 examples; in the other 3 examples this unit was the 25th, i.e., a 6th lumbar.

Willis quotes Le Double,⁴ who, in addition to describing 11 similar cases, gives a summary of the examples of the same condition reported in the literature. Apparently congenital division of the lamina has been described in the 1st lumbar and the 1st sacral vertebrae on one occasion each, in the 4th lumbar twice, and in the 5th lumbar on 29 occasions. The greatest incidence of congenital division of the lamina evidently is on the 5th lumbar vertebra, the normal last pre-sacral unit.

In the series of specimens from Bantu natives described in this paper, congenital division of the lamina has been found in six vertebrae contained in five vertebral columns. In three skeletons the last pre-sacral vertebra was affected, in one the penultimate, and in one the last two pre-sacral vertebrae. The high incidence of this developmental defect in the skeletons of Bantu natives is noteworthy. The five columns which exhibited congenital division of the lamina were included in a series of 56 unselected skeletons.

From such a comparatively small number, it would not be possible to represent the proportion of Bantu skeletons exhibiting congenital division of the lamina as 5 out of 56, or as 9.1 per cent, but it may be stated that these abnormalities were found in a systematic examination of all the Bantu skeletons in the department for another purpose, and that no selection whatever has been exercised.

SUMMARY.

1. An anomalous mode of development of the lower lumbar vertebrae occasionally results in the separation of the dorsal arch by a congenital cleft in the laminae.

2. In American white and negro races this phenomenon occurs in 4.28 per cent of vertebral columns, in the Bantu races of South Africa the proportion seems to be higher.

3. The condition of separation of the dorsal arch of a vertebra due to congenital division of the laminae may be antecedent to the condition of spondylolisthesis.

It seems probable that the dorsal arch, completely separable in the dried state, in *Specimens II, III, and IV*, in life was connected to the rest of the vertebra by cartilage, or more likely by fibrous tissue.

The factors which bring about separation and dislocation of the vertebral body must be a matter of conjecture. Trauma must certainly be suspected. Perhaps a disease of ligamentous tissues, similar to that which precedes the occurrence of flat-foot in certain cases of gonorrhoea, might prepare the way for dislocation as a result of minimal violence.

Other causes of forward dislocation of a vertebral body suggest themselves—for example, caries of the body of the 1st sacral or the 5th lumbar units, or fracture of the vertebral arch. Nevertheless, it seems that dislocation of the body of the 5th lumbar vertebra associated with separation of the dorsal arch, as described by Ziegler and quoted above, is most readily explained by the anomalous development of the lower lumbar vertebrae of which examples have been given in this paper.

I have to make grateful acknowledgements, first to Professor Raymond A. Dart, for placing the material at my disposal; and secondly to Mr. E. L. Nolan to whose skill and care I am indebted for the photographs.

REFERENCES.

- ¹ ZIEGLER, *Textbook of Special Pathological Anatomy*, 1898, section iv, 229.
- ² THOMPSON, PETER, *Morris's Treatise on Anatomy*, 5th ed., part 1, 48.
- ³ WILLIS, T. A., "The Lumbo-sacral Column in Man, its Stability of Form and Function", *Amer. Jour. of Anat.*, xxxii, 95.
- ⁴ LE DOUBLE, *Traité des Variations de la Colonne vertébrale de l'Homme*, quoted by Willis.

THE ORIGIN OF ACOUSTIC NERVE TUMOURS.*

BY H. ALAN SKINNER,

DEPARTMENT OF ANATOMY, UNIVERSITY OF TORONTO.

THE many and varied contributions to the subject of acoustic nerve tumour during the last fifty years offer a complexity and divergence of observation and opinion that have endowed the question of the origin of these tumours with peculiar interest for the investigator, not only from the standpoint of the tumour itself, but also in the general field of nerve pathology.

The most outstanding contributions appear to be those of Henschen¹ and Cushing.² In two articles, "Ueber die Geschwülste der hinteren Schädelgrube, insbesondere des Kleinhirnbrückenwinkels", and "Zur Histologie und Pathogenese der Kleinhirnbrückenwinkeltumoren", Henschen carefully reports his findings as regards pathology and histology in a large series of cases. Cushing's

book, *Tumors of the Nervus Acusticus*, which appeared in 1917, embraces a wider field, including clinical aspects and surgical procedure, with a review of the various pathological features and findings. In general Cushing appears to agree with Henschen's conclusions.

According to Henschen and other investigators the acoustic nerve consists of two distinct segments which differ from each other histologically. This applies to both divisions of the nerve. These segments of the nerve are designated as central and peripheral. The central segment is also referred to as the glial portion and the peripheral segment as the non-glial portion. The glial portion has the histological characters of fibre tracts within the brain substance—that is to say, the endoneurium and neurilemma sheath (sheath of Schwann) are



FIG. 358.—Henschen's reconstruction of an early acoustic nerve tumour.

missing, and the supporting tissue consists of glia-cells. In the peripheral (non-glial) portion the endoneurium and neurilemma sheath are present, and this segment therefore presents the appearance of a typical peripheral nerve. The structure of the nerve will be more fully discussed later.

* This paper represents the result of work done through the subvention of The Banting Research Foundation.

Careful investigation of the available literature reveals nothing that is contradictory to Henschen's statement that the tumour originates in the distal (peripheral, non-glial) part of the vestibular division of the eighth nerve. The most important confirmatory evidence of this assertion is the fact that all the small tumours, which have been more or less accidentally found, have obviously been associated with the vestibular division of the nerve. The most interesting and well investigated early tumour is that described by Henschen, of which he made a reconstruction in wax, the illustration of which is reproduced here (*Fig. 358*). The facial nerve is seen lying alongside the tumour, the cochlear nerve is almost entirely hidden by the growth, while the vestibular nerve is seen to spread out at the central end of the mass, embrace the substance of the tumour, and then gradually disappear. At the distal end vestibular fibres again appear and pass off the tumour into a distal trunk.

In the face of the evidence on this point, one can hardly deny the fact that the starting-point of tumours of the acoustic nerve appears to be on the vestibular division of the nerve. Henschen's second point, that the tumour invariably occurs on the distal (non-glial) segment of the vestibular nerve, also appears to be well substantiated, and will be further referred to later.

Having regard to these findings, several interesting problems are presented. First, what cell which normally occurs in the peripheral segment of the vestibular division of the auditory nerve is the type cell of the tumour? Second, what is the normal structure of this nerve? Third, why is this tumour comparatively common on this nerve and exceedingly rare, if not unknown, on other cranial nerves?

In order to answer these questions, and particularly the last one, a clear understanding of the development and normal structure of the eighth cranial nerve is necessary. The first consideration, therefore, is that of some of the embryological characters of this region.

THE DEVELOPMENT OF THE EIGHTH CRANIAL NERVE.

The various steps in the formation of the auditory vesicle will not be detailed here. This aspect of the embryology of the region has been well worked out by Streeter, and does not appear to play an important part in the elucidation of the present problem. Henschen, however, notes in this connection that the eighth cranial nerve differs somewhat from the other nerves in the fact that its peripheral portions are largely enclosed in a bony capsule by the developing petrous bone, which he states is not entirely developed from pre-formed cartilage but attains its final form through the later ossification of connective tissue. He notes this condition at the bottom of the meatus, where the auditory nerve comes in closest relation to the bone.

The formation and development of the acustico-facial ganglion complex is a problem which still appears unsettled. In some of the lower forms which have been investigated from the standpoint of the development of the cranial ganglia, it would appear that certain contributions to the ganglion masses are made from ectodermal placodes. Knouff³ reports that the acustico-facial ganglion mass of the frog receives a contribution from the ventromesial wall

of the auditory vesicle. The same author also credits Streeter with the statement that this ganglion mass is evidently not derived from the neural crest in man. He also quotes Giglio-Tos as describing in man lateral and epibranchial contributions. Adehnann,⁴ reporting on the cranial ganglia of the rat, states that the auditory ganglion is a derivative of the common acustico-facial ganglion mass, owing its origin entirely to neural crest proliferation. W. His, jun.,⁵ reporting on the development of the acustico-facial region in man, observed no placodal contributions. Also Weigner,⁶ in a similar communication, was unable to detect any connection of the ganglion mass with the ectoderm.

The form of the ganglion mass is characteristic. It early assumes a triangular or trefoil shape which is an indication of the eventual splitting. Its position is ventral and slightly medial to the auditory vesicle. The facial portion can be distinguished at an early stage by the pale-staining character of its cells. It is also noticed that the cells of this portion are the largest.



FIG. 359.—Section from 6.0 mm. human embryo showing acustico-facial ganglion complex and auditory vesicle.



FIG. 360.—Section from the opposite side of the embryo of Fig. 359, showing appearance of vestibular fibres.



FIG. 361.—Section from 9.0 mm. human embryo showing relation of ganglion mass to vesicle.

For some time the remainder of the ganglion mass remains undivided. Eventually the dorso-lateral portion differentiates off as the vestibular ganglion, while the remaining dorso-medial part results in the cochlear ganglion. The mass of cells, at first separated by a thick layer of mesoderm from the auditory vesicle, gradually approaches it, and the first indication of fibres appears in the sections which were available for study by the author. These sections were of human embryos, and three photographs (Figs. 359-361) are reproduced to show the form, appearance, and relation of the ganglion mass to the vesicle in human embryos of 6.0 and 9.0 mm. Fig. 359 shows the typical arrangement and the relations of ganglion mass and vesicle. They are still separated by a distinct band of mesoderm. The pale-staining ventral portion of the mass is the *Anlage* of the geniculate ganglion. In Fig. 360 the appearance of fibres is noted. These are from the dorso-lateral

portion of the mass and therefore are undoubtedly vestibular fibres. In *Fig. 361* a still closer connection of this portion of the ganglion mass with the vesicle is shown, while there is still no evidence of cochlear fibres.

When the cochlear fibres have also made their connections it is apparent that the relations of the two dorsal portions of the ganglion mass (vestibular and cochlear portions) have changed. The vestibular ganglion has become more dorsal, the cochlear ganglion more ventral. At the same time the lateral (vestibular) portion is associated with the medial auditory root, while the medial (cochlear) portion is associated with the lateral root (His). This arrangement results in a crossing of the fibres. To illustrate this condition there is in *Fig. 362* a copy of the semi-diagrammatic illustration of W. His, jun., which shows the arrangement at this stage. The relative positions of the ganglia and the course of their fibres are easily seen. One result of this shift in position is that the cochlear nerve is lengthened while the vestibular ganglion is brought nearer the brain stem. The cochlear nerve is further lengthened at a later period by the twisting which it suffers in the formation of the spiral whorls of the cochlea, so that eventually the nerve-cells of the spiral ganglion come to be much farther removed from the brain-stem than those of Scarpa's ganglion.

The crossing of the two divisions is not usually apparent in adult human sections, as one seldom obtains a block of tissue sufficiently large to include the entire nerve. In some of the smaller animals, however, the normal arrangement can be determined, and in sections of rabbit heads prepared during this investigation the crossing of the two divisions of the eighth cranial nerve was quite apparent.

The question of the central connections of the fibres brings up a new factor. This is the appearance of the sheath-cells. From the work of Harrison⁷ it appears that the neurilemma-cells have their origin in the ganglion mass and are neural crest derivatives. At the same time, the entire length of the eighth nerve does not exhibit a sheath of Schwann (neurilemma), but in its central segment is glial in type, the glia replacing the endoneurium as well as the neurilemma sheath. The glia-cells which are present in the central segment of the nerve must have migrated from the brain-tube. It is extremely probable that the cells observed by Neal⁸ were glial cells in the act of migrating. It is further evident that no such migration of glia can occur until the central connections of the fibres are completed. It also seems probable that the fibres are functional before ensheathing begins.

With regard to the migration of glia along the nerve-fibres, it is interesting to note that the vestibular fibres complete their connections before the cochlear fibres. It therefore appears reasonable to assume that migration of glia begins in the vestibular nerve at an earlier date than in the cochlear. As for the other ensheathing elements, there is no reason to assume that there is any

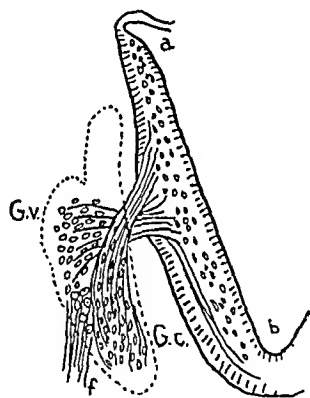


FIG. 362.—Copy of semi-diagrammatic illustration from Wm. His, jun. a—b, Brain tube; G.v., Vestibular ganglion; G.c., Cochlear ganglion; f, Facial nerve.

difference in the date of beginning ensheathment of fibres by neurilemma-cells.

The work of Harrison shows that the neurilemma-cells originate in the ganglion masses as neural crest derivatives. The cells of the neural crest, therefore, give rise to at least two types of cell, the ganglion-cell and the sheath-cell. Therefore the elementary, uniform, neural-crest cell must eventually differentiate into two different types. These appear to pass through several stages. In the case of the ganglion-cells associated with the eighth cranial nerve it is interesting to note that these cells do not develop as far from the embryonic type as the cells in other ganglia; in other words, they remain as bipolar cells throughout life. On the other hand, there is no reason to suppose that the specialization of the sheath-cells is any different in this region from that in any other. So that it appears possible that the ganglion-cells develop their final form, send out processes, and make their connections before any sheath-cells have developed to the point where they

are capable of assuming their final rôle. A simple scheme of differentiation is indicated in *Fig. 363*.

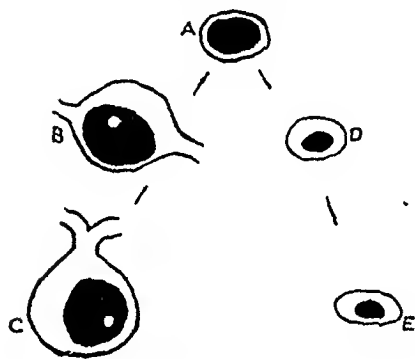


FIG. 363.—Diagram to illustrate the differentiation of nerve-cells and sheath-cells. A, Neural-crest cell; B, Bipolar nerve-cell; C, Unipolar nerve-cell; D, Neurilemmablast; E, Neurilemma sheath cell.

The result of this condition appears to be as follows: The vestibular fibres make their connections. The glial outgrowth into the central end of the vestibular division commences. Later the cochlear fibres make their connections. The glial outgrowth into these fibres then commences. Still later, the sheath-cells originating in the ganglion mass (neurilemma-cells) commence to grow centrally. It is therefore apparent that the glia of the vestibular nerve will progress further peripherally than the glial outgrowth into the cochlear nerve

before encountering the peripheral ensheathing cells, which meeting takes place at a considerable distance from the brain-stem.

The occurrence of glia in the central segment was remarked by Henschen and investigated as to its extent. His results were as follows: "The total length of the auditory nerve in an adult male amounts to 17 to 19 mm., in a female 16 to 17 mm., in a seven-month child, 15 mm. The apex of the glial cup in an adult male lies 10 to 13 mm. outside the middle cerebellar peduncle, in the female 7 to 10 mm. The border on one side can lie more distal than the border on the other. Sometimes the boundary lies at a somewhat different level in the two branches of VIII, when it is likely to be somewhat more distal in the vestibularis."

Fig. 364 is reproduced from Henschen's article. It shows the two segments of the vestibular nerve. At -v is situated the vestibular ganglion. The glial portion is plainly shown as the paler section in distinction to the darker, peripheral part.

Other investigators have determined the presence of a glial part, and

Hulles (*see* Henschen) puts the point of junction only 5 mm. from the brain. Henschen's figures, however, seem to be more accurate. These place the point of junction near the plane of the porus and this appears to be correct, although one would think that the plane of the internal acoustic meatus would represent the most distal point to which the glia ever attains. This is a point, however, which requires corroboration. Henschen and others mention glial outgrowths in the nerve beyond this boundary. An indication of these is shown in *Fig. 364*. A few light streaks or patches are seen in the darker, peripheral segment. These presumably are glial islands, but it is possible that these so-called glial islands are only so in appearance, and on closer examination and by other staining methods will reveal themselves in other wise. This, too, is a point which requires corroboration.

The investigation of the cranial nerves by means of gold and silver stains should throw very considerable light on this question. As we are more concerned here with the peripheral part of the auditory nerve, it is not intended to include any such investigation in this present paper, except to present *Figs. 365* and *366*, which are included to elaborate the text. They are preparations made by the gold sublimate method and photographed under low power. *Fig. 365* is a section of the seventh cranial nerve, showing a normal peripheral nerve with this stain. Note the clear, orderly,



Fig. 364.—Section of vestibular nerve, with facial nerve to left. To show glial and non-glial parts. -V, Vestibular ganglion. (Henschen.)



Fig. 365.—Facial nerve. Gold sublimate stain. (Low power.)



Fig. 366.—Auditory nerve. Gold sublimate stain. (Low power.)

parallel arrangement of the fibres. *Fig. 366* is a section of the auditory nerve taken close to the point of emergence from the brain. Note the apparent lack of arrangement and the spotting of the section with astrocytes.

Summary of Development of the Auditory Nerve.—

1. The ganglion mass appears to be developed entirely from the neural crest.
2. The vestibular fibres appear before the cochlear fibres.
3. The final position of the ganglion mass is such that the vestibular nerve is shorter than the cochlear.
4. Both branches of the eighth nerve show comparatively little lengthening in the course of their development. They are the shortest nerves in the body.
5. The cells of both ganglia (vestibular and cochlear) retain the comparatively primitive bipolar character.
6. There is a glial outgrowth from the brain over the central segments of both cochlear and vestibular divisions.
7. This outgrowth extends farther peripherally on the vestibular division than on the cochlear.
8. The level of the glial boundary may be different on the two sides of the same individual.

THE NORMAL STRUCTURE OF THE AUDITORY NERVE.

In this section only the peripheral (non-glial) portion of the nerve will be considered. The peripheral portion of the auditory nerve is, in general,

very similar to any other peripheral nerve. It possesses axones which have a sheath of myelin, with further sheaths of neurilemma-cells and endoneurium, the whole surrounded by epi- and perineurium.

There are, however, certain features of the auditory nerve which are somewhat different from the ordinary type of peripheral nerve, particularly so in the region of the vestibular ganglion.

The first point observed in examining this nerve under the microscope was that it appeared to be very heavy in



FIG. 367.—Human auditory nerve, region of vestibular ganglion. To show connective tissue elements and scattering of ganglion-cells. (Low power.)

its connective-tissue components. The connective tissue of the peri- and epineurium is prominent, and more or less splits the nerve into bundles. A cross-section of the nerve in the region of the vestibular ganglion illustrates this fact (*Fig. 367*). Preobraschensky also notes this point (Hensehen): "At the peripheral side of this nerve the connective tissue is markedly increased . . .". This unusual amount of connective tissue normally present in the auditory nerve is of considerable interest in the present investigation.

When one examines the nerve under higher magnification it is apparent that the endoneurium is also involved in the increase in connective tissue. The number of fibroblasts is usually excessive, and at first sight the nerve presents something of the cellular picture of an embryonic nerve. Most of these cells can be identified as fibroblasts in the trunk of the nerve. The other cell type present, the neurilemma-cell, is not so easy to identify. Many nuclei, which fall on the borderline between the two types, are difficult to place in the absence of any other method of determination beyond shape and staining characters.

Fig. 368 is a section of the eighth cranial nerve (human) stained with methylene blue. A number of fibroblast nuclei are easily distinguishable. One small, oval, but nearly round, nucleus is visible. This nucleus takes the stain quite deeply in contrast to the fibroblast nuclei, whose chromatin is rather finely divided and easily seen. The small nucleus appears to be a fairly dense mass of chromatin. In addition to this nucleus three borderline nuclei appear in the field. Two of these are out of focus. They are small, rather darkly stained, yet difficult to place, although they may easily be neurilemmal nuclei. The one which is closest to the small, dark nucleus appears from its position to lie within the endoneurium of the nerve, which would confirm the assumption of its neurilemmal character.

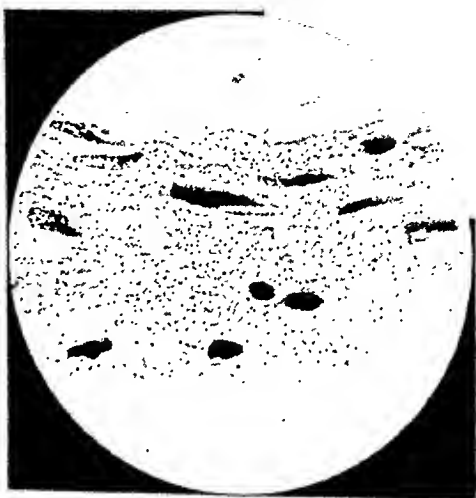


FIG. 368.—Normal auditory nerve. Adult human. Methylene-blue stain, oil-immersion lens.



FIG. 369.—Normal auditory nerve. Adult human. Van Gieson's stain, oil-immersion lens.

Fig. 369 is a similar section of an adult human eighth nerve, but stained with van Gieson's stain. The fibres of the connective-tissue cells are now apparent. Once again it is possible to distinguish several fibroblast nuclei without difficulty, while there are several smaller nuclei which are more difficult. The two nuclei indicated in the photograph are picked out as interesting. The long, spindle-shaped nucleus is obviously a connective-tissue nucleus, and indeed its fibres can be seen, tapering off from each end. The smaller nucleus lying to the right.

Note that this fibre line passes around the smaller nucleus lying to the right.

This latter nucleus, while still oval, is more nearly round. These two cells, it is believed, represent the two supporting elements of peripheral nerve, the neurilemma-sheath and the connective tissue. The numerical relation between them appears to be, normally, about six to one in favour of the fibroblast.

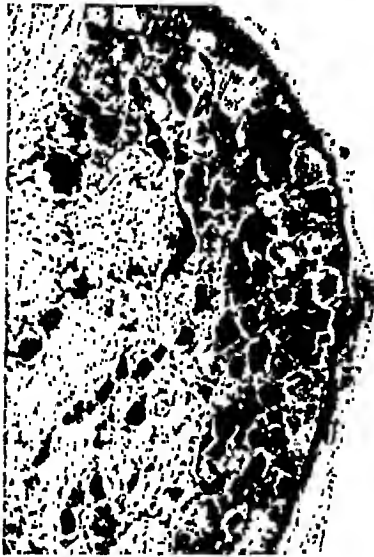


FIG. 370.—Low-power view of dorsal-root ganglion of cat.

When one comes to examine the ganglia that are associated with this nerve the problem becomes more complex. The questions at once arise, What is the normal structure of any ganglion? What is the relation of the sheaths of the nerve to the sheath of the ganglion-cell? What are the so-called subcapsular cells of the ganglion?

In the normal dorsal-root ganglion the ganglion-cells appear to arrange themselves most compactly around the periphery, forming a horseshoe-shaped arrangement, between the points of which the nerve-fibres emerge. In the central part there is a mixture of axone-fibres and nerve-cells, but around the periphery the ganglion-cells are close together and present the appearance

of a closely packed mass of cells in which it is difficult to make out any nerve filaments. This arrangement is illustrated by *Fig. 370*, which is a low-power view of a section of a dorsal-root ganglion of a cat.



FIG. 371.—Low-power view of spiral ganglion of rabbit.



FIG. 372.—Low-power view of geniculate ganglion of rabbit.

A similar arrangement of nuclei and fibres is seen in the cochlear ganglion. In the various parts of the spiral one sees massed ganglion-cells with a central core of nerve-fibres extending out into the 'shelf' toward the organ of Corti. The disposition of nerve-cells and fibres here is comparable to that of the dorsal-root ganglion. *Fig. 371* is a photograph (low power) of the spiral ganglion of a rabbit. The arrangement is similar in the human.

In the case of ganglia which occur on the trunk of a nerve, such as the geniculate ganglion of the facial and the vestibular ganglion, the arrangement is somewhat different. *Fig. 372* shows a section of the geniculate ganglion of the rabbit. The ganglion-cells tend to clump together, but in no definite order. The nerve-fibres are visible, coursing among the cells. They tend to isolate small masses of ganglion-cells, in which, however, the ganglion-cells themselves cling close together with very little intervening tissue. In the vestibular ganglion the scattering of ganglion-cells is more pronounced. The



FIG. 373.—Dorsal-root ganglion of cat.
Van Gieson stain, oil-immersion lens.



FIG. 374.—Same as *Fig. 373*, showing
part of fibrous capsule.

nerve-cells tend to take a more isolated situation, being irregularly scattered through a wider area. *Fig. 367*, which is a low-power view of a vestibular nerve section, illustrates this feature. The scattering is not, however, always as prominent as in this section, where it is especially marked.

Figs. 373 and *374* are views of the dorsal-root ganglion taken through the oil-immersion lens. The so-called subcapsular cells are at once evident. They are found lying about the circumference of the ganglion-cells and in close relation to them. Fine connective-tissue fibrils can be distinguished in these sections, lying in between the ganglion-cells. The subcapsular cells are found occupying a place between these fibrils and the ganglion-cells; in other words, they have the same relative position as the neurilemma-cell occupies in the nerve-trunk. It appears probable that these subcapsular cells

are the same cell as the neurilemma-cell and that the ganglion-cell has, in fact, a double sheath—an outer one composed of fibroblasts and their processes, and an inner one directly continuous with the neurilemma-sheath of the nerve.

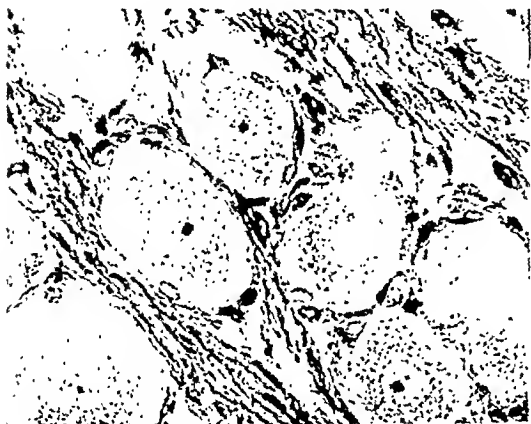


FIG. 375.—Geniculate ganglion of rabbit. Van Gieson stain, oil-immersion lens.

The size, shape, and general appearance of these subcapsular nuclei is exactly similar to that of the neurilemma nucleus.

One also observes in these sections the close proximity of the ganglion-cells to each other. There is very little intercellular tissue. In *Fig. 374* a portion of the capsule of the ganglion is shown. This consists of a number of layers of connective-tissue cells in which it is possible to distinguish typical fibroblast nuclei. This capsule runs in an orderly way around the ganglion mass, enclosing it in a fibrous sheath which is directly contin-

uous with the perineurium. It is also noteworthy that there is no extensive migration of fibroblasts into the interior of the ganglion. Here and there a fibroblast nucleus can be identified, and between the ganglion-cells the fine

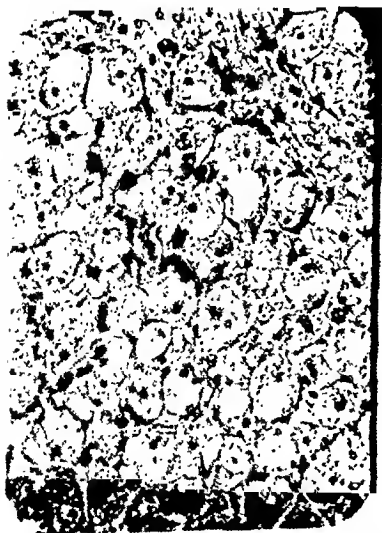


FIG. 376.—Cochlear ganglion from human foetus of seven months. Van Gieson stain, high dry lens.

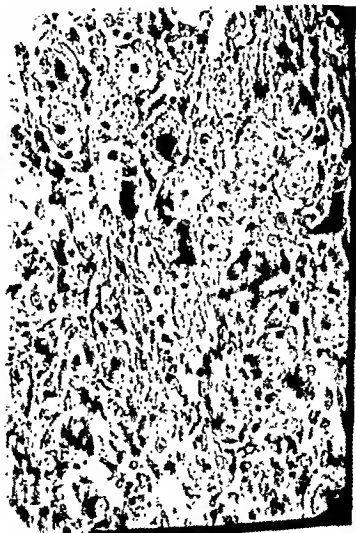


FIG. 377.—Same case as *Fig. 376*. Vestibular ganglion. Van Gieson stain, high dry lens.

fibrils mentioned above are evident. Nowhere is there seen anything in the nature of heavy fibrous bands or trabeculae.

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In the geniculate ganglion of the rabbit (*Fig. 375*) under the oil-immersion lens the same general scheme is apparent, although here the picture is somewhat different on account of the numerous nerve-fibres which traverse the region. The subcapsular cells are readily identified, there is the same difficulty in recognizing any marked connective-tissue structure, and the relation of ganglion-cells to each other is similar in the various areas where they tend to clump.

One now comes to a consideration of the vestibular ganglion, and as the cochlear division of the eighth nerve is closely associated with the whole question which we are pursuing, its ganglion will be figured at the same time for comparison. *Figs. 376 and 377* are two photographs taken with the high dry objective. *Fig. 376* is from the cochlear ganglion of a seven-month foetus, *Fig. 377* shows the vestibular ganglion of the same subject.

In the cochlear ganglion the characteristic features already pointed out are again seen. The ganglion-cells form a compact mass in which each individual cell is in close relation with the adjacent ganglion-cells. Inter-cellular substance is not present to any great extent. Nerve-fibres are not distinguishable except at the upper end of the section, where the beginning of a collection of fibres is visible.

In the vestibular ganglion a very different picture is presented. The ganglion-cells are much more scattered, and numbers of nerve-fibres course through the field. The entire area is spotted with nuclei which cannot be identified at this magnification. In comparison with the corresponding cochlear ganglion this area presents an irregular disordered appearance. In comparison with the geniculate ganglion, which presents a closer parallel as regards structure than the cochlear, the principal impression of difference is in the more cellular nature of the tissues lying between the ganglion-cells of Scarpa's ganglion.

The next two photographs show the cochlear ganglion (*Fig. 378*) and the vestibular ganglion (*Fig. 379*) under the oil-immersion lens. The differences previously pointed out are accentuated and confirmed.

In the cochlear ganglion the massed arrangement is still evident. The ganglion-cells are in close relation with each other, the intercellular tissues are scanty. Occasional nuclei are visible between the ganglion-cells, and these nuclei present the same characters of form and staining as the subcapsular nuclei observed in the dorsal-root ganglion, and are of similar size, shape, and staining character as the neurilemmal nuclei seen in the nerve-trunk.

In the vestibular ganglion the differences are now very marked. Ganglion-cells are widely separated. The most striking observation to be made in this section is the very cellular nature of the tissues lying between the ganglion-cells. Large numbers of nuclei are apparent. Again, the majority of these present characters typical of subcapsular cells of other ganglia and of the neurilemmal nuclei. The significant fact here, however, is the lack of any kind of orderly arrangement of these nuclei. Whereas in other ganglia the subcapsular cells were definitely arranged in an orderly way about the circumference of the ganglion-cell, now they appear in a jumbled-up, irregularly concentrated, meaningless state of disorder. One would have difficulty in ascribing a functional rôle to each of the irregularly placed nuclei occurring

in this confused accumulation. That a certain number are occupying positions definitely related to the ganglion-cells cannot be denied. The number that can be so placed, however, corresponds approximately to the number of nuclei of similar nature seen in other ganglia. In addition to these more or less normally situated nuclei there are at least double the number of irregularly placed nuclei which occupy the areas intervening between the ganglion-cells.

Some of the nuclei seen in *Fig. 379* are undoubtedly fibroblast nuclei, but the number that can be so identified is very small. It seems to be more than probable that the great majority of these excess nuclei are not of a fibroblastic nature, but, as their appearance and staining reactions suggest, are closely related to the neurilemmal nuclei and the subcapsular cells.

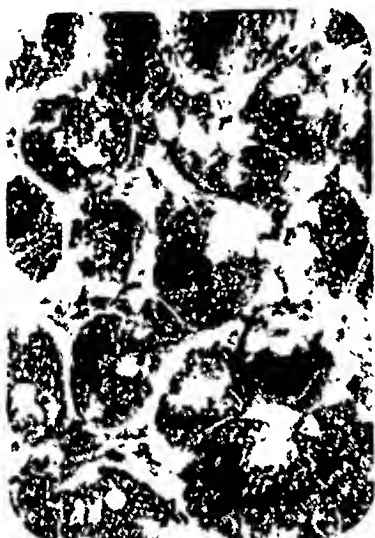


FIG. 378.—Cochlear ganglion from human foetus of seven months. Same as *Fig. 376*. Oil-immersion lens.



FIG. 379.—Same as *Fig. 377*. Vestibular ganglion seen with oil-immersion lens. Van Gieson stain.

In the human adult is reproduced the condition as pictured here in the foetus. Exactly the same conditions have been found during this investigation in the cellular nature of the human adult vestibular ganglion.

What is the explanation of the occurrence of these nuclei in this situation? One satisfactory answer presents itself. As has been seen, in the development of this nerve the central connection is made at an early date, earlier than in the case of the cochlear nerve. On this account the glial outgrowth has been able, in the case of the vestibular division, to proceed to a greater distance from the brain-stem, with a resulting diminution in the remaining portion of unsheathed nerve; so that, when the sheath-cells developing from the ganglion *Anlage*—that is to say, the neurilemma-cells—become differentiated and ready to assume their ensheathing rôle, a comparatively short section of nerve remains unenclosed by the neuroglia. This condition is accentuated by the shorter course of the vestibular nerve. Such explanation is in accord with Henschen's findings, where, if we may repeat,

it is stated: "The apex of the glial cup in an adult male lies 10 to 13 mm. outside the middle cerebellar peduncle, in the female 7 to 10 mm. The border on one side can lie more distal than the border on the other. Sometimes the boundary lies at a somewhat different level in the two branches of VIII. when it is likely to be somewhat more distal in the vestibularis." It might also be noted that Hensehen's findings in the case of small infants are in the same proportion as the findings in adults. For example: "5-month-old female, length of VIII, 15 mm.; glial part 7 mm., non-glial 8 mm."

The situation appears to be this. In the case of the vestibular division of the eighth cranial nerve the ganglion *Anlage* differentiates into ganglion-cells and sheath-cells. The number of sheath-cells is sufficient for the ensheathing of a certain indefinite distance of nerve-fibre. In the case of other ganglia this distance is considerable. But in the vestibular nerve this distance is short, and it has been further considerably decreased by (1) the early completion by the vestibular fibres of their peripheral and central connections, permitting an extensive ensheathment at the central end by outgrowing glia-cells, and (2) the comparatively late differentiation of the neurilemma-cells, the peripheral ensheathing element, which also permits the glia-cells to reach further peripherally. When one says that the neurilemma-cells are comparatively late, one refers to the ganglion-cells that remain in the bipolar stage which represents an incomplete differentiation or development (*see Fig. 363*). Therefore a considerable number of neurilemma-cells seems to be left over and to lie in their primitive position between the cells of Scarpa's ganglion.

Summary of the Normal Structure of the Auditory Nerve.—

1. The structure of the peripheral portions of the nerve-fibres is similar to other peripheral nerves. One recognizes axone-fibres, neurilemma-cells, and connective tissue.
2. There appears to be an increased amount of fibrous tissue present in the peripheral portion of this nerve.
3. The ganglion-cells of the cochlear ganglion present an arrangement similar to that observed in other ganglia.
4. The cochlear ganglion presents a normal arrangement of tissues occurring in relation to the ganglion-cells.
5. The ganglion-cells of the vestibular ganglion are very scattered; they occupy no regular position or arrangement in relation one to the other.
6. The cells lying in the regions between the ganglion-cells of the vestibular ganglion are considerably increased in amount; they occupy no regular position or relationship to the ganglion-cells and present a disordered appearance.
7. These irregular cells resemble neurilemma-cells and the subcapsular cells observed in other ganglia.

THE TUMOUR OF THE ACOUSTIC NERVE.

It is not intended here to enter into the clinical side of this condition, although one or two points of clinical interest will be brought out. This part of the problem has been carefully investigated by Cushing, and the reader is referred to his work for clinical details that are omitted here.

The next step in the investigation is the examination of tumour material. For the purposes of this paper five tumours were examined. For the majority of these we are indebted to Dr. K. G. McKenzie, of the Surgical Department of this University, whose assistance has been greatly appreciated. In all cases where it was possible to obtain fresh tumour material in the operating room, portions of tumour were placed in formol-bromide solution for examination by the gold sublimate and silver carbonate methods. In all cases a complete examination of all material by the ordinary staining methods was carried out.

The gross appearance of these tumours is not always identical; in fact two groups are distinguished clinically, which division is more or less borne out by the microscopic examination. One group consists of large, lobulated, knobby tumours, which when cut into are tough and fibrous, bleed freely, and often appear cystic in places. The other group as a rule consists of rather smaller tumours, which more resemble the white matter of the brain in their cut surface appearance and consistency; they are more easily removed piecemeal, and do not bleed so extensively.

The first impression on examining these two groups is that one is dealing with two different tumours, one of which suggests a glioma, the other a fibroma. This would fit in perfectly with the two parts of the auditory nerve, and also with the divergent opinions of different authors. The two types, however, are not so well marked off. The tumour varies from one extreme to the other without any essential change in the character of its components. What these components are is a subject of prime importance.

When one considers the variety of terms which appear in the literature in description of the cell type of this tumour, one anticipates some difficulty in arriving at a determination of the type cell. While it is our intention to present only two of the five cases examined by us, it will, one thinks, suffice, as the other three cases present no findings which are not apparent in the two given, and also a careful investigation of the reported cases, where detailed descriptions of the microscopic findings appear, should convince one that all these tumours have the same essential characters.

The first case had a typical history of some four years' duration. The patient underwent an operation and a portion of the growth was removed. It was one of the tough, fibrous tumours, and considerable bleeding was encountered, so that a complete removal was inadvisable if not impossible. About six weeks after the operation the patient died. The material presented here was obtained at autopsy, and consists of a large piece of tumour with which is bound up a portion of the eighth cranial nerve.

Fig. 380 is a view of a cross-section of the entire mass. This photograph is about double the actual size. It was made from a celloidin section stained with hæmatoxylin and eosin. At the right of the section the nerve is seen curving into the substance of the tumour mass. The knobby, lobulated appearance of the tumour is suggested by the uneven outline of the section. Large cystic areas are visible, into some of which hæmorrhage has taken place. The upper, central portion of the tumour, however, presents little or no evidence of degeneration and appears to be a solid mass of tumour tissue.

When one examines this area closely it presents a mottled or marbled appearance. This character can be directly correlated with the low-power pictures of the area. It is from this undegenerated region that the photographs of this tumour were made.

Before going on to the examination of the tumour itself, a view of the nerve as it enters the mass will be of interest. *Fig. 381* is a photograph of the nerve under the oil-immersion lens. A few small dark-staining nuclei are visible, but the majority are undoubtedly fibroblastic; more fibroblasts are present than in the normal nerve. The nerve tissue here, too, seems to have a more fibrous appearance than

that of normal nerve, suggesting a replacement of nerve fibrils by fibrous tissue. Somewhat deeper in the substance of the tumour it is impossible to recognize anything like nervous tissue. The fibres shown here simply spread out in all directions and are soon lost in the tumour mass. The appearance presented by the nerve in this section is that of a brisk fibroblastic reaction.

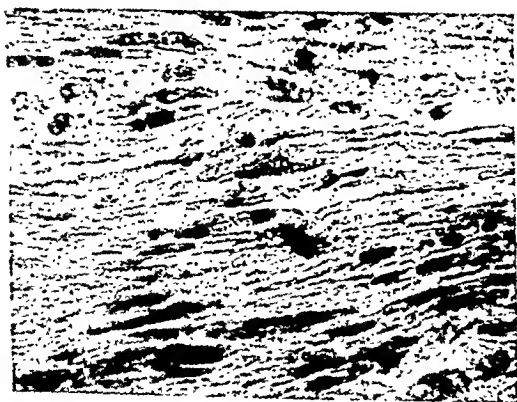


FIG. 381.—Auditory nerve entering tumour. Van Gieson stain, oil-immersion lens.



FIG. 380.—Acoustic nerve tumour. Haematoxylin and eosin stain. ($\times 2$.)

On examining with the low power an area where no degeneration has taken place, the first impression is one of fibrous tissue. This is more or less the typical picture presented in many contributions to the pathology of this condition, and descriptions of fibrous areas with a palisade arrangement of nuclei are so general that this palisading has almost become the essential point in diagnosis of tumours of the acoustic nerve. Penfield,⁹ in a recent article, stresses this palisaded arrangement and the general disposition of the fibrous tissue.

If, however, one examines the tissue more carefully, it is apparent that there is another type of cell occurring all through the substance of the tumour.

In some areas, where the fibrous bands are particularly heavy, it is almost impossible to distinguish anything but fibroblasts; in other areas, where the



FIG. 382.—Acoustic nerve tumour. Typical area under low power. Hematoxylin and eosin stain.

fibrous tissue is not so dense, one sees a variable number of smaller nuclei taking a more or less prominent part in the picture, depending on their concentration in any one region. In *Fig. 382* there is a general view of the average sort of tissue found in a typical area of an acoustic tumour of the fibrous type. Bands of fibrous tissue are a prominent feature. The nuclei associated with these bands are obviously fibroblastic in appearance, and will be more clearly shown with a higher magnification. In addition to this frank fibrous tissue one also sees, scattered through the field, smaller dark nuclei, in varying numbers. It is difficult to distinguish at this magnification what sort of

nuclei these are, the low power being entirely inadequate for determination of type. The mottled or marbled appearance, referred to earlier in the description, is explained by the veining of the cut surface by fibrous bands, which appear light in contrast to the darker areas of nuclear concentration. In *Fig. 383* another area is shown. Here an accumulation of small dark-staining cells is located in one place, forming a sort of nest, cut off by heavy fibrous bands. Note the compact crowded nature of this nest of cells and compare with low-power views of the second case. It is not difficult to imagine such an island, choked off by the heavy fibrous tissue, undergoing some kind of degeneration. This type of picture is frequently seen on looking over preparations of this tumour. It is also noticed (*see Fig. 380*) that this tumour has suffered considerable degeneration in some areas, particularly cystic degeneration. It is probable that

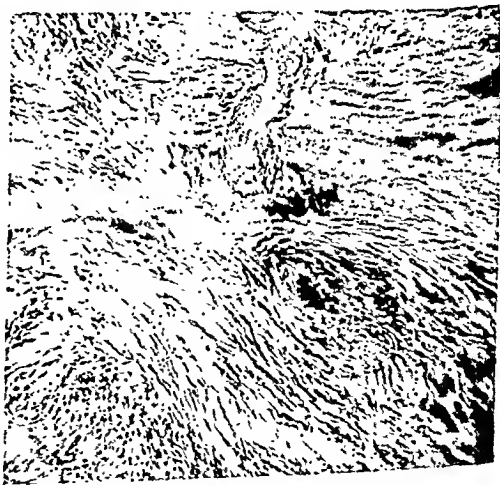


FIG. 383.—Acoustic nerve tumour under low power. Area of tumour nest. Hematoxylin and eosin stain.

these nests of cells, their circulation cut off by the marked fibrosis, are the points where necrosis first takes place and degeneration sets in.

Examination of the tissues with a higher magnification indicates more precisely the nature of the cells present. In a typical fibrous area, in one of the fibrous bands, a picture as shown in *Fig. 384* is presented. Associated with these bands are nuclei which are definitely fibroblastic. The nuclei are elongated in the direction of the fibres; the chromatin is not massed, but more or less thinly dispersed throughout the nucleus. The length of the nucleus is usually many times greater than its breadth, and the definite association of this type of nucleus with fibrous tissue and its absence in other situations confirms its identity. When we turn to an area such as presented in *Fig. 383*, where there is a nest or accumulation of smaller dark cells, or to an area lying between two fibrous bands, an area which might be referred to as a cellular area in distinction to a fibrous area, we find a different picture (*Fig. 385*). Here is a collection of nuclei which bear no resemblance whatever to fibroblasts. They are small, oval, but nearly round, and only one or two of the many nuclei in the field suggest that they might be fibroblasts cut obliquely. No fibres can be distinguished. The cells appear to be arranged in no kind of order, being in all sorts of positions and occupying every possible relation to one another. That this is a second type of cell is evident. It is also evident that these cells occur in no sort of ordered arrangement but occupy a position far removed from such—in fact the position of tumour-cells. On looking over the section one repeatedly identifies these two types. The fibroblast nuclei are always definitely associated with fibrous areas. Where fibrous processes cannot be distinguished, one finds the smaller type of nucleus.

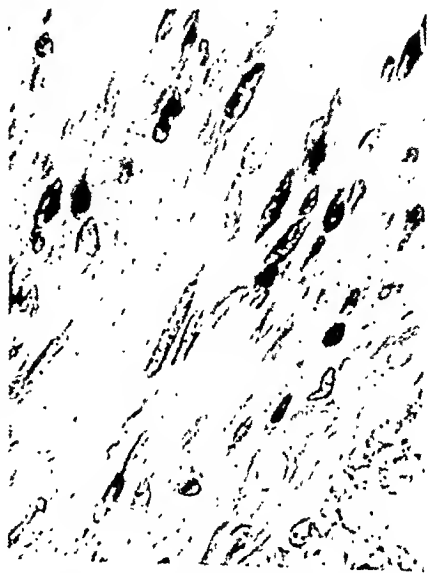


FIG. 384.—Acoustic nerve tumour. Fibrous band under oil-immersion lens. Van Gieson stain.



FIG. 385.—Acoustic nerve tumour. Cellular area under oil-immersion lens. Van Gieson stain.

If one compares the size of these two types of nuclei, one is immediately reminded of the comparison in size between the fibroblasts of the normal

nerve on the one hand and the neurilemma nuclei and subcapsular nuclei on the other. If one then compares the smaller cells of the tumour with the cells which are seen so abundantly in the region of the vestibular ganglion, one immediately sees that these two have nuclei of the same shape, size, and staining characters.

The second specimen illustrates the conditions in the other type of tumour—the softer tumour which resembles the white matter of the brain. A section of the entire tumour cannot be shown, as the material was obtained at operation and consists of pieces of tumour which vary in size from that of a small pea to a bean. This material was ‘spooned out’ by the operator. The tissue is fairly uniform in consistence and presents no cystic areas or other macroscopic evidence of degeneration. It is a pale pinkish-white colour which takes a slight yellowish-brown tinge on fixation in formalin. The usual staining technique was carried out on pieces which were fixed in formalin and in Zenker’s fluid. Other pieces were placed in formol-bromide for examination for neuroglia.

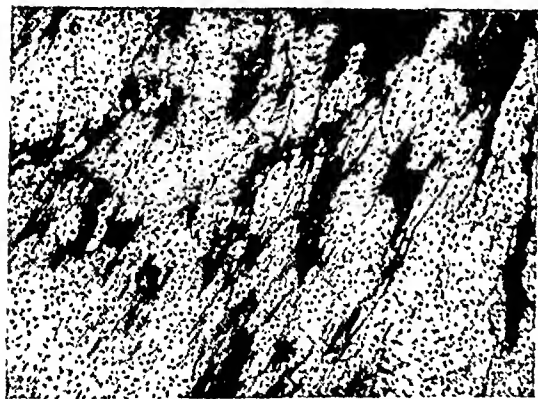


FIG. 386.—Low-power view of acoustic tumour (second type). Weigert stain.

In sections stained with van Gieson’s stain a general fine fibrous network can be made out. This is readily seen with the low power, and between the strands of the network one sees, generously distributed throughout the section in a more or less uniform fashion, cells which have a small nucleus. The striking difference between the low-power views of the two cases is the absence of the heavy

fibrous bands in this second case, a circumstance of some significance when considered in the light of the fact that this second case presents no evidence of degenerated areas.

An excellent idea of the relation of fibrous tissue and cellular areas is obtained from a low-power view of a section stained by the Pal-Weigert method. It also emphasizes the very striking nature of the picture as seen under the low power of the microscope. Fibrous areas occur irregularly here and there throughout the section, but scattered everywhere are the smaller nuclei, which stand out clearly and distinctly in the light areas where the fibrous tissue appears to be missing (*Fig. 386*).

It is also noteworthy that these small cells which are so plentiful here are nowhere compressed together into anything resembling a cell-nest, such as was seen in the first case. This fact, along with the previously mentioned difference in the fibrous content and the lack of degenerated areas in this second case, appears to point out the process which results in the production of cystic and other degenerated areas.

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In a high-power view of this section stained by the Pal-Weigert method the nuclei can be more clearly seen and the distinction between the two types confirmed. With this stain the fibrous tissue shows darkly, while the non-fibrous areas are clear. *Fig. 387* is a high-power (dry) photograph of a typical area of the second case stained by this method. All nuclei which, from their shape, can be identified as fibroblasts are definitely associated with the dark fibrous streaks. On the other hand, the smaller nuclei appear constantly in the clear areas where fibroblasts are never seen and where there is no indication of fibrous tissue. This appearance is reproduced by other staining methods.

This tumour was also investigated from the standpoint of neuroglia content. All sections stained for neuroglia-cells and processes were negative, as was the case in one other tumour so examined.

There seems to be no positive evidence that neuroglia plays any part in the production of this tumour, not only from this present investigation but also from a careful examination

of the literature on this question. Taken along with the results of Henschen's investigation, where he asserts definitely that in no case was any direct connection of the glial part of the nerve with the tumour made out, one can with reasonable surety affirm that this tumour is not a glioma.

In the investigation of tumour material from five cases no ganglion cells have been identified in the sections. Nor has there been any evidence of a growth of nerve-fibres such as one sees in an amputation neuroma. It is always possible that an occasional ganglion cell may turn up in the examination of one of these tumours, but there should be no hesitation in stating that these tumours are not neuromas.

The only remaining possibilities that are at all likely are the connective tissue and the neurilemma sheath of the peripheral portion of the nerve. In fact it appears to be the general opinion that one of these cells is the type cell of this tumour, with a tendency to label it a fibroblastoma. In the light of the present investigation it is the author's opinion that the neurilemma sheath cell (or its precursor, the 'neurilemmablast') is the type cell of the tumour, and that the large amount of connective tissue which is so often present in such amount as to make a diagnosis of fibroma (or fibroblastoma) frequent, can be regarded as a reaction to the growing tumour-cells in an attempt to wall them off and restrict the untoward growth. It is interesting in this connection to note the fact that the probable origin of the neurilemma-cells is from the neural crest—that is to say, from the ectoderm—

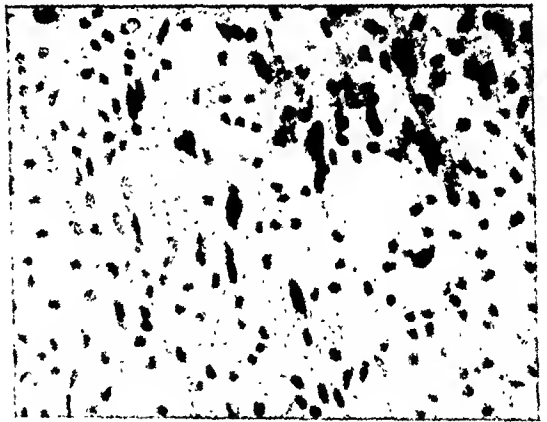


FIG. 387.—Dry high-power view of acoustic tumour (second type). Weigert stain.

while the fibroblast is a mesodermal product. Trotter's¹⁰ view of a conflict or reaction between the two tissues is of interest on this point.

In regard to the two types of tumour described, one point of interest which would be worthy of further investigation is the relation of the type of tumour to the clinical picture. The very fibrous type with degeneration processes might well be associated with a different type of history from that of the more cellular type. In the two cases given here the distinction between them as regards clinical history is slight, but it nevertheless seems to be a point that is of practical importance. In this connection it is worthy of note that the more cellular type of tumour is much more easily handled at operation, as the fibrous type tends to bleed when torn and is also more liable to be adherent to dura and other tissues. With regard to adherence to surrounding tissues there is considerable evidence in the literature, particularly in Hensehen's series. Unfortunately one cannot determine accurately from the description of the histological findings in the reported cases what was the relation of fibrous tissue to the second type of cell. It is thus difficult in most cases to determine whether any particular tumour should be classed as a fibrous or a cellular type. It is also probable that many tumours occupy an intermediate position between the two types. The possibility, however, that the fibrous type represents a slow-growing tumour, well walled off by fibrous tissue, is a point of sufficient importance to be investigated in the future, as it would be of considerable interest clinically to determine from the history and symptoms, if possible, whether the tumour were fibrous or cellular before the case came to operation.

In nearly all the cases reported by various authors the two cell types—a fibroblast and a smaller, rounder cell—are noted. The smaller cell is usually interpreted as glial, although many possibilities are advanced. The fibrous tissue, however, always receives more attention, with the result that the most common diagnoses vary from fibroma to neurofibroma and fibrosarcoma.

The areas occupied by the smaller type of cell are frequently referred to as oedematous areas. This assumption is probably due to the distribution of the nuclei, which frequently are rather more widely separated than one would expect. These regions are, however, solid, and in the cellular type, where they are most common, one does not find any frank oedematous areas. It seems more probable that the apparent space between the nuclei is taken up by the cytoplasm of the cells. In the normal nerve there appears to be only one neurilemma-nucleus to each segment of the nerve-fibre. The length of one segment (between two consecutive nodes of Ranvier) varies from 0.1 to 1.1 mm. One presumes, therefore, that one neurilemma-cell is able to provide a thin sheath for a considerable length of nerve-fibre. If a neurilemma-cell is then removed from its functional situation and allowed to resume the spherical form, it is evident that the cytoplasm will occupy a considerable space. This also appears of interest in respect to the scattered ganglion-cells of the vestibular ganglion, and suggests that the bulky 'neurilemmablasts' filling in the area between ganglion-cells are largely responsible for this scattering. It is also interesting to observe the effect of the isolation of a group of tumour cells by heavy fibrous tissue such as is shown in *Fig. 383*.

Here the nuclei are very close together, suggesting a compression and disappearance of the cytoplasm, the nuclei being the last portions to degenerate.

If one accepts the neurilemma-cell as the type cell of the acoustic nerve tumour, the question of some other nerve tumours becomes of interest. It is well known, for instance, that bilateral acoustic nerve tumours are frequently associated with von Recklinghausen's disease, generalized neurofibromatosis. It seems very probable to the author that this condition may also reveal itself as the same type of tumour formation as occurs on the eight nerve. There is, of course, no evidence to this effect in the present contribution. The hypothesis, however, presents some interesting possibilities. It is suggested that all these tumours are closely bound up with the question of the ensheathing of nerve-fibres. The ensheathing of peripheral nerves seems to involve two processes. The first is the gradual extension of the neurilemma sheath along the nerve-fibre. The second suggests itself as a reaction of the mesoderm to the cells of the sheath of Schwann. Under certain conditions naked nerve-fibres have been observed to occur in mesodermal surroundings without producing any marked reaction. Generally speaking, however, when there is a sheath of neurilemma-cells present there is always produced an endoneurium formed of fibroblasts. One would think that the neurilemma-cells presented an especial stimulus or irritation to the mesoderm, so that a second ensheathment or walling off the neurilemma-cells takes place. Is there any evidence to support such a contention? Leaving out the evidence of tumours such as are dealt with here, which in itself is not sufficient evidence, there is one fact that seems very significant. This is the behaviour of the neurilemma-cells following nerve section. It is a matter of frequent observation that in the degenerating part of the nerve there is considerable evidence of activity on the part of the neurilemma-cells. This is principally exhibited as an enlargement or swelling of the whole cell, with some change in its form. This would suggest an inherent quality or potentiality of these cells to revert to a more embryonic state, which condition would appear to be bound up with the growth and regeneration of nerve tissue. The supposition that seems natural is that these cells possess the power to increase in size, divide if necessary, and take up an active proliferating rôle in contrast to a normal, passive, ensheathing function. Is this potentiality of the cells the irritant quality that causes the ubiquitous fibroblast to wrap the nerve round with an extra outer sheath? If one could suppose such a condition, one could easily visualize, in the case of generalized neurofibromatosis, the neurilemma-cells escaping from the ensheathing fibroblasts, which cells in turn would hasten to surround the proliferating neurilemma-cells. If this condition repeated itself frequently, as might easily be the case were the neurilemma-cells particularly potent, or the fibroblasts generally unequal to their task, then the ease with which multiple tumours would spring up on the course of nerves is apparent. That the primary cause of generalized neurofibromatosis lies in a hyperplastic activity of the neurilemma-cells is somewhat borne out by the fact that other disturbances in cells of ectodermal origin are usually apparent in the form of pigmentation of the skin, moles, abnormal growths of hair, and other manifestations.

It seems distinctly probable that the neurilemma-cell is the type cell in

a large group of tumours. With regard to the particular problem here, it is apparent that if these cells have the *potentiality* of growth and increase barely restrained at all times, and ready to appear quickly as seen in the case of regenerating nerve, then such a situation as has been shown to be present in the region of the vestibular ganglion must always present a distinct possibility for the production of a tumour. The action of the fibroblasts is also thereby explained, and the final form and character of the tumour is only an indication of the ability or inability of the fibrous tissue to cope with the neurilemma-cells. The fact that the peripheral part of the eighth nerve is particularly heavy in fibrous tissue is another indication that this tissue is called out wherever there is present the stimulus of potent neurilemma-cells.

One more side to the clinical problem may be reiterated. A point which has not received the prominence it deserves is the fact that the vestibular nerve is the starting-point of the tumour. Most of the clinical histories record symptoms associated with the cochlear nerve, but careful investigation of the vestibular reactions does not appear to be a common procedure. Many cases have been remarked in which the hearing was good until a short time before operation, which is not surprising if the vestibular nerve is the primary seat of the disease. In this connection it is interesting to note in Cushing's historical review a case reported in 1810, where a woman of 38 had primary vertigo, followed by headache, loss of vision, and then deafness in the left ear. The importance of the point is that the vestibular reactions in suspicious cases is of primary interest and may yield valuable information on the subject of early diagnosis.

GENERAL SUMMARY AND CONCLUSIONS.

1. Acoustic nerve tumours arise on the vestibular division of the eighth cranial nerve, usually distal to the plane of the porus acusticus internus.
2. The type cell of the tumour is the neurilemma sheath cell.
3. The central, glial segment of the nerve is never primarily involved in the tumour.
4. The somewhat exact position of their origin in the peripheral segment is dependent on peculiar factors in the embryological development of this nerve.
5. The fibrous tissue, often present in large amounts, is in the nature of a tissue reaction.
6. This reaction between neurilemma-cells and fibrous tissue is normally observed under various circumstances.
7. The tumours may be divided into two types, a cellular type and a fibrous type.
8. The type is determined by the ability or inability of the fibroblasts to confine the tumour-cells.

The author wishes to express his thanks to Professor J. P. McMurrich for his advice and assistance, to Dr. E. A. Linell for his active co-operation and constant help throughout this entire investigation, and to Dr. Mary Tom for her valuable assistance in technical matters.

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REFERENCES.

- ¹ HENSCHEN, FOLKE, "Zur Histologie und Pathogenese der Kleinhirnbrückenwinkel-tumoren", *Arch. f. Psychiat.*, lvi.
- ² CUSHING, HARVEY, *Tumors of the Nervus Acusticus and the Syndrome of the Cerebello-pontine Angle*, 1917, Philadelphia: W. B. Saunders Co.
- ³ KNOUFF, R. A., "Cranial Ganglia of Rana", *Jour. of Comp. Neurol.*, 1927, xliv, 259.
- ⁴ ADELMANN, HOWARD B., "Neural Folds and Cranial Ganglia of Rat", *Ibid.*, 1925, xxxix, 19.
- ⁵ HIS, WILHELM, JR., "Zur Entwicklungsgeschichte des Acustico-Facialgebietes beim Menschen", *Arch. f. Anat. u. Physiol.*, 1889, Supp.
- ⁶ WEIGNER, K., "Bemerkungen zur Entwicklung des Ganglion acustico-faciale und des Ganglion semilunare", *Anat. Anzeig.*, 1901, xix.
- ⁷ HARRISON, R. G., "Further Experiments on the Development of Peripheral Nerves", *Amer. Jour. Anat.*, 1906, v, 121.
- ⁸ NEAL, H. V., *The Development of the Ventral Nerves in Selachii*, Mark Anniversary Volume, New York, 1903.
- ⁹ PENFIELD, WILDER, "The Encapsulated Tumours of the Nervous System", *Surg. Gynecol. and Obst.*, 1927, Aug., xlv, No. 2.
- ¹⁰ TROTTER, WILFRED, "The Insulation of the Nervous System", *Lancet*, 1926, ii, 107.

TUBERCULOUS BACILLURIA :
ITS INCIDENCE AND SIGNIFICANCE AMONGST PATIENTS
SUFFERING FROM SURGICAL TUBERCULOSIS.

By R. I. HARRIS,
 ASSISTANT SURGEON TO THE HOSPITAL FOR SICK CHILDREN, TORONTO.

It has not been generally recognized that tubercle bacilli may be recovered with remarkable frequency from the urine of patients suffering from bone and joint tuberculosis. From time to time reports have appeared in the literature which record the incidence of tubercle bacilli in the urine of patients suffering from tuberculosis (usually pulmonary tuberculosis). Such examinations have revealed a percentage of positive urines, which, while not great, was unexpected, since they occurred in patients who presented no symptoms of renal tuberculosis. These symptomless cases of tuberculous bacilluria have usually been interpreted as evidence of the excretion of tubercle bacilli by the kidney, though such a conclusion is based upon slight foundation. The present investigation not only demonstrates the frequent occurrence of tuberculous bacilluria amongst surgical patients (as high as 37 per cent), but indicates that the tubercle bacilli probably originate in foci of renal tuberculosis. As far as I have been able to ascertain, the demonstration of such a high incidence of renal tuberculosis amongst tuberculous patients is a new observation, and since it throws some light upon the difficult pathology of renal tuberculosis, it is of sufficient importance to merit careful scrutiny.

For a proper conception of the importance and significance of tuberculous bacilluria amongst surgical patients it is necessary to consider briefly the pathology of surgical tuberculosis. The course of events leading to the inception of foci of bone tuberculosis can best be studied in children, since in them the disease has existed for a relatively short time and the picture is not confused by secondary extensions of the disease or exacerbations of old foci. The great majority of such patients are infected from human cases of open pulmonary tuberculosis, and as the infection is air-borne the common primary focus is in the lung. Such a primary pulmonary lesion is small. It is of importance, not only because of the damage it produces in the lung, but also because it is a focus from which tuberculosis may be disseminated to other parts of the body. Tubercle bacilli readily leave the primary focus and pass in the lymph-stream to the nearest lymph gland. There they set up a fresh focus of tuberculosis. From this secondary lesion they again pass in the lymph-stream to the next gland in the chain. By a series of such steps the disease progresses until the whole of the lymphatic chain draining the area of the primary focus is the seat of tuberculous adenitis—mediastinal tuberculous adenitis when the primary focus is in the lung. It is likely that

most primary pulmonary lesions terminate at this stage by a slow increase in resistance which permits the defensive mechanism of the body to stamp out the infection. Healing at such a stage probably represents the type of tuberculosis from which the majority of us have unconsciously suffered.

In certain cases, however, the disease progresses. From the mass of tuberculous mediastinal glands, tubercle bacilli reach the blood-stream by way of the thoracic duct. Usually they do so in relatively small numbers, and no doubt, under ordinary circumstances, these scattered invaders of the blood-stream are effectively disposed of by phagocytosis in various organs of the body. Under certain conditions, however, a single organism or a group of organisms may gain a foothold in distant tissues and there set up a fresh focus of tuberculosis remote from the primary lesion. In this fashion single or multiple isolated foci arise in any part of the body.

Although it has no important bearing upon the inception of foci of bone tuberculosis, mention should be made of the facts that the body may be invaded at other points than the lung and that the spread from the primary focus may be along other pathways than the lymphatics. Primary foci may be implanted in the tonsil or in the intestinal mucosa as well as in the lung. Two or more primary foci may be present at the same time; in all probability this is more common than is suspected; at any rate it frequently can be demonstrated that patients have, for example, primary foci in both lung and tonsil. As is the case in the lung, the common path of spread from tonsillar and intestinal foci is by way of the lymphatics—and results in tuberculous cervical adenitis and tuberculous mesenteric adenitis. While lymphatic spread is the most common occurrence, the blood-stream may be directly invaded by rupture of a caseous focus into an adjacent vessel. In the lung the primary focus may rupture into a bronchus and be disseminated to all parts of the bronchial tree, giving rise to tuberculous bronchopneumonia. In the etiology of bone tuberculosis the lymph-stream is the important means of spread, since the other pathways tend to produce such massive infections (tuberculous bronchopneumonia, miliary tuberculosis, or tuberculous meningitis) that death rapidly ensues. The common primary foci and their common paths of spread may be represented diagrammatically as in *Fig. 388*.

Among the most common of the hæmatogenous foci whose origin has been described are those lesions of bones and joints which constitute surgical tuberculosis. The hæmatogenous nature of joint tuberculosis does not appear to have been sufficiently stressed. At any rate the conception commonly exists that tuberculosis of a joint is tuberculosis of a joint and nothing more. Such, of course, is not the case. Their hæmatogenous nature makes it evident that every such lesion must have arisen from some pre-existing primary focus. It is commonly true that the primary focus is inconspicuous and symptomless, while the bone lesion is evident and the source of many signs and symptoms; nevertheless, the existence of tuberculosis in a bone predicates the existence of at least one other focus of tuberculosis from which it has arisen after invasion of the blood-stream.

Of even more importance for our present purpose is the evident fact that if tubercle bacilli have been present in the blood-stream in numbers sufficient to produce one focus of hæmatogenous origin, it is possible and

even probable that other hæmatogenous lesions may have been laid down. This is indeed the case. One has only to examine a number of patients with bone and joint tuberculosis to discover that many of them have multiple lesions. In a series of 100 patients whom I have examined for this purpose, more than half, on adequate examination, displayed multiple foci of hæmatogenous origin. Some years ago Peabody,¹ in studying a series of cases of Pott's disease, was able to demonstrate that certain of them presented multiple separate foci of Pott's disease in the same spine.

Renal tuberculosis originates by infection of the kidney from the blood-stream. It would not be unreasonable, therefore, to expect to find this tuberculous lesion more commonly amongst patients who have bone tuberculosis

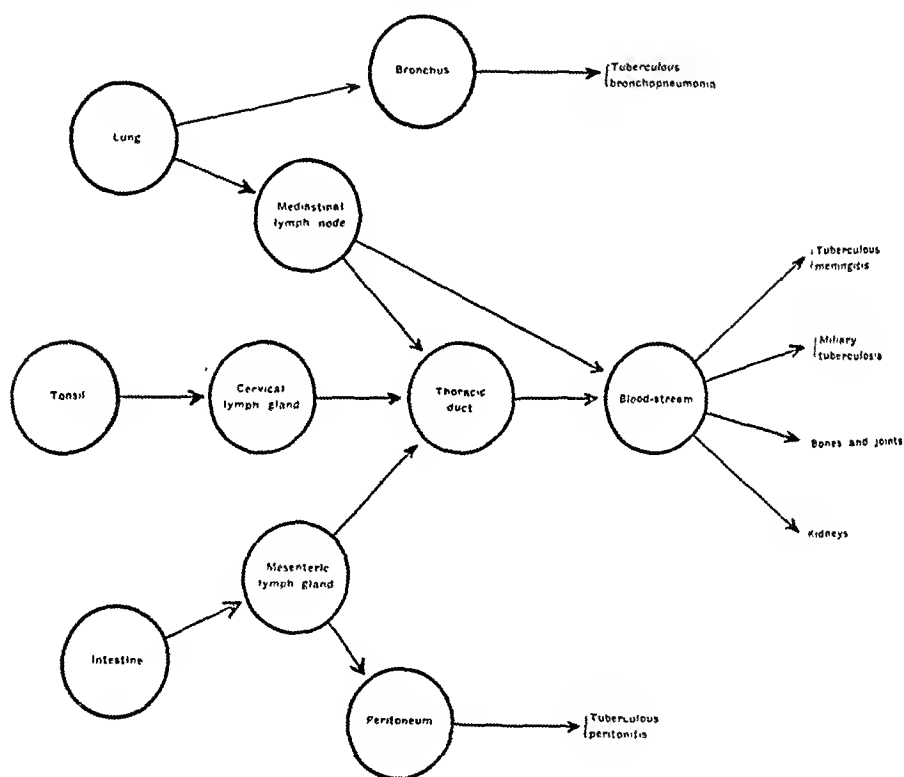


FIG. 388.—Diagrammatic representation of the primary foci and of the paths of invasion in tuberculosis.

than amongst those who have only pulmonary tuberculosis. In the former the blood-stream has been invaded, and the intensity of the tuberculous septicæmia is evidenced by the presence of hæmatogenous foci. In the latter the disease is still limited to the seat of the primary focus; if the blood-stream has been invaded, there is no conspicuous evidence of that fact. The conception of the etiology of bone tuberculosis which has been outlined gives an added significance, therefore, to the occurrence of renal tuberculosis in patients who have bone lesions. For these reasons a case of renal tuberculosis arising in one of my surgical patients aroused my interest. The

known difficulty in diagnosing early renal tuberculosis made it seem possible that careful examination of a number of patients would reveal evidence of renal tuberculosis in some who presented no obvious symptoms. Such an investigation was carried out, and the results form the basis of the present paper. The investigation consisted in the careful examination of the urine from a considerable number of patients suffering from bone and joint tuberculosis. Urine was collected under sterile precautions into a sterile receptacle. Part of it was examined chemically and microscopically, and the sediment from the remainder was inoculated into a guinea-pig. When the report of the guinea-pig inoculation was received at the end of two months, a second specimen was collected and dealt with in a similar manner. A series of such tests was carried out at intervals of two months as long as the patient was under observation, so that there is available for most of the patients a long series of urinalyses extending over periods of eighteen months or two years. The first patients were all adults, but later the series was expanded to include a number of children.

In all there have been examined by this method 43 adult patients and 67 children. Some of the former have been observed over five years; in the majority, however, the period of observation has been two or three years. The juvenile cases are more recent, since work on them has been carried out only during the past eighteen months. Of the adult cases, 16 (37 per cent) were found to have tubercle bacilli in the urine, intermittently or constantly. Amongst the children, 9 (13·8 per cent) were similarly positive. For purposes of comparison similar observations were carried out on a number of tuberculous patients without bone and joint involvement. Amongst these patients (49 in number), 4 (8·8 per cent) had tubercle bacilli in the urine. All four positive cases occurred in patients in whom gross invasion of the blood-stream was evidenced by the presence of miliary tuberculosis or tuberculous meningitis. Condensed reports of all the positive cases follow.

ADULT CASES WITH BONE TUBERCULOSIS.

Case 1.—M. L., male, age 25, admitted to hospital in September, 1922, without any evident primary focus, but multiple hæmatogenous lesions (lumbar spine, right hip, rib, and breast), suddenly developed mild renal symptoms accompanied by pyuria. For a year tubercle bacilli could not be found in the urine, but since then (a period of nearly four years) they have been present constantly. The renal symptoms have subsided and at the present time are inconspicuous. Cystoscopy has revealed findings which have been interpreted as indicating bilateral renal tuberculosis. The outcome is still in doubt, but it seems likely that extensive renal disease is present, probably bilateral, and that in the course of time more conspicuous evidences of renal tuberculosis will appear.

Case 2.—P. G., male, age 32, was admitted in September, 1925, suffering from tuberculous cervical adenitis and a spinal lesion of hæmatogenous origin. The patient had pus constantly, and tubercle bacilli occasionally, present in his urine, though symptoms of renal involvement were never present during the whole period of observation. The renal origin of the tubercle bacilli was not proved. Under treatment directed to his Pott's disease he made an apparent cure, in so far as the presence of tubercle bacilli in the urine was concerned. Pyuria was still present on discharge from hospital.

Case 3.—E. C., male, age 32, with old pulmonary tuberculosis and a hæmatogenous lesion in his spine, developed a transient tuberculous bacilluria coincident with the appearance of a tuberculous epididymitis. The significance of this finding is open to question. The tuberculous bacilluria may have been secondary to the tuberculous lesion in the testis and seminal vesicles, or the lesions in the external genitals may have arisen as the result of the presence of tubercle bacilli in the urine, in a manner similar to that in which gonorrhœal epididymitis arises.

Case 4.—N. S., male, age 28, with tuberculous cervical adenitis and a hæmatogenous lesion in the spine, developed a transient tuberculous bacilluria without gross pyuria and without symptoms of renal tuberculosis. It is likely that a small renal lesion was present and that this later underwent spontaneous healing.

Case 5.—H. R., male, age 29, with old pulmonary tuberculosis and a hæmatogenous lesion in the spine, developed a transient pyuria and tuberculous bacilluria. The significance of this is open to question. The presence of pus suggests that the tubercle bacilli came from a tuberculous focus in the kidney. The duration of the pyuria and tuberculous bacilluria is not known, since the adjacent samples were two months before, and six months after, that which contained tubercle bacilli.

Case 6.—H. B., male, age 29, with pulmonary tuberculosis, a hæmatogenous lesion in the spine, and old genital tuberculosis, had tubercle bacilli and small amounts of pus constantly present in the urine. The significance of this is open to question, since cystoscopy has not been performed.

Case 7.—E. H., male, age 35, with pulmonary tuberculosis and multiple hæmatogenous lesions (elbow, rib, and tarsus) and with genital tuberculosis, had pus and tubercle bacilli constantly present in his urine during three years of observation. At no time during this period have any renal symptoms been present. The renal origin of the organisms was definitely demonstrated by cystoscopy.

Case 8.—E. D., male, age 27, admitted to hospital in September, 1925, with tuberculous cervical adenitis and a hæmatogenous focus in the spine, had tubercle bacilli constantly present in the urine from the time he first came under observation. Pus has been present in very small amounts. Frequency at first was not increased, but now is slightly greater than normal. Even at the present time his symptoms are so slight that they might easily escape notice. Tubercle bacilli have been demonstrated in the urine from both kidneys. The outcome of this renal infection is still in doubt. Its steady though slow progress would seem to indicate that he ultimately will develop the usual features of bilateral renal disease.

Case 9.—T. F., male, age 36, with pulmonary and gastro-intestinal tuberculosis and a hæmatogenous lesion in his knee without evident renal symptoms, developed tuberculous bacilluria coincident with a slight though definite increase in the amount of pus present in his urine. While cystoscopy has not yet been performed it is reasonably certain that the tubercle bacilli are of renal origin, since the genitals are free from foci of tuberculosis. Routine examination of the urine permitted the early detection of the disease. Ordinarily this would not have occurred until symptoms arose. The outcome is in doubt, though the intensity of his tuberculous infection, and his poor defence against it, make steady progress more likely than cure.

Case 10.—G. S., male, age 30, admitted in May, 1927, with old pulmonary tuberculosis and a hæmatogenous lesion in the spine, through the early part of three years of observation had a few pus cells constantly present in the urine, and tubercle bacilli occasionally. During the whole of this time he presented no symptoms of renal tuberculosis, nor has he since. He seems in good health and has apparently recovered from his renal tuberculosis.

Case 11.—R. S., male, age 46, admitted in August, 1926, with pulmonary tuberculosis, a hæmatogenous lesion in his shoulder, and evidences of involvement

of the genito-urinary tract (tuberculous epididymitis). He had constant pyuria and tuberculous bacilluria, though without the usual symptoms of renal tuberculosis. The renal origin of at least some of the tubercle bacilli was demonstrated by cystoscopy. Post-mortem examination revealed no tuberculosis of the right kidney, but many small lesions in the left kidney, well advanced in healing. His most active lesion was in the bladder. My impression is that this patient's genito-urinary infection commenced in the kidney, with later extensions to the bladder and epididymis. The renal lesions remained small and progressed towards healing, while the vesical lesion became more extensive.

Case 12.—W. P., male, age 33, admitted to hospital in October, 1924, without any evident primary focus but with a hæmatogenous lesion in the spine, presented some symptoms of renal involvement in the form of mild-frequency. The urine contained pus and tubercle bacilli on admission. The frequency, pyuria, and tuberculous bacilluria disappeared under rest and heliotherapy, and he has remained free from symptoms of renal tuberculosis since his discharge. The discovery of tubercle bacilli in the urine from the right kidney demonstrates their renal origin and precludes the possibility that they might have been derived from the tuberculous epididymitis. In addition, tuberculous bacilluria was present at least ten months before the lesion in the epididymis appeared. In my opinion this is an example of healed, or apparently healed, renal tuberculosis.

Case 13.—P. C., male, age 27, admitted to hospital in December, 1921, with pulmonary tuberculosis, developed numerous hæmatogenous foci over a period of six years. Exclusive of the genito-urinary lesions, no fewer than seven of these hæmatogenous foci were identified. During this period of intense infection of the blood-stream, tubercle bacilli were found in the urine. Their presence was at first unaccompanied by any of the usual evidences of renal involvement, but these later gradually appeared, until the classic symptoms of renal tuberculosis were present in great-severity. The lesion has steadily progressed and a fatal termination seems certain.

Case 14.—S. N., male, age 26, with pulmonary tuberculosis and a hæmatogenous lesion in the spine. On admission to hospital he presented the classic signs and symptoms of renal tuberculosis. Cystoscopic examination demonstrated the lesion to be renal and bilateral. The symptoms steadily increased up to the time of his death. Post-mortem examination demonstrated the presence of bilateral renal tuberculosis, though the renal lesions were microscopic in size. The disease was much more active and extensive in the bladder. My impression is that during a period of heavy blood-stream invasion the kidneys were heavily infected with innumerable foci of the miliary type. From the kidneys the bladder was infected. The renal lesions did not progress; on the contrary, there was some attempt at healing. The vesical tuberculosis increased and produced the greater part of his symptoms.

Case 15.—E. K., male, age 28. In childhood the patient had passed through a severe illness, which was probably a generalized tuberculosis. At the time of his admission to hospital in August, 1922, he had old pulmonary fibrosis and pleuritis, a hæmatogenous lesion in his spine, and tuberculosis of the testes; upon routine examination of the urine he was found to have a mild pyuria and a persistent tuberculous bacilluria. These findings persisted for a year unaccompanied by symptoms. The urinary findings steadily increased in severity, and there gradually appeared the symptoms of renal tuberculosis. At the time of his death he presented the classic picture of renal tuberculosis. Post-mortem examination revealed the complete destruction of the left kidney by a caseating tuberculous process (auto-nephrectomy) and pyonephritis of the right kidney.

Case 16.—R. M. C., male, age 37, without any evident primary focus, but with multiple hæmatogenous lesions, developed the typical symptoms of renal tuberculosis. Removal of the grossly infected kidney in December, 1926, rapidly resulted in an

apparent cure, in spite of the fact that tubercle bacilli were recovered from the remaining kidney. Fifteen months later the passage of large quantities of pus from the remaining kidney indicated that a considerable lesion was present in it. The remarkable feature about this case is the long period of latency during which no symptoms were present.

Table I.—RESULTS OF ANALYSIS OF URINE IN ADULT CASES OF BONE TUBERCULOSIS.

	Urine Negative	Urine Positive
Pott's disease	17	12
Tuberculous knee	4	1
Tuberculous shoulder	1	1
Tuberculous hip.. .. .	3	1
Tuberculous elbow	2	1
	<hr/> 27	<hr/> 16 (37 per cent)

JUVENILE CASES OF BONE TUBERCULOSIS.

Case 17.—A. Z., a girl, age 3½, admitted to hospital in May, 1927, with a probable primary focus in the lung and a hæmatogenous focus in the hip, showed a transient tuberculous bacilluria unaccompanied by pyuria or symptoms. The significance of this is open to question, since pus cells were not found in the urine. It is probably of importance to note that the single positive specimen was obtained early in her illness, when she was suffering severely from the infection.

Case 18.—J. S., a boy, age 12, without any evident primary pulmonary lesion, but with two hæmatogenous foci in the spine, displayed an intermittent pyuria and tuberculous bacilluria, but unaccompanied by any symptoms of renal tuberculosis. This case probably represents a minimal renal lesion undergoing spontaneous cure, though definite confirmation by positive ureteral specimens is lacking.

Case 19.—H. S., a girl, age 11, admitted in May, 1922, without evident primary focus, but with two hæmatogenous foci in hip and knee. The patient has displayed a constant pyuria and intermittent tuberculous bacilluria for five years. During this period there have been no symptoms of renal tuberculosis. It has been impossible to collect ureteral specimens because of the changes in the bladder mucosa. This case probably represents a silent renal infection, whose outcome is still debatable. The last positive specimen was obtained two and a half years ago. The enlarged right kidney probably indicates disease on that side at least. I should think it possible that the disease might undergo spontaneous cure or that it might light up into renewed activity.

Case 20.—T. F., a boy, age 11, without any evident primary focus, but with a hæmatogenous lesion in his hip. The patient was admitted to hospital in December, 1925, and found to have a constant mild pyuria and tuberculous bacilluria. It was possible by cystoscopy to demonstrate that the tubercle bacilli came at least from the left kidney. During the period of observation frequency developed, so that he now presents in a mild degree the classic picture of renal tuberculosis. Even at this date the symptoms are so mild that it is certain they would be overlooked were not special attention paid to the urine. This patient represents a renal infection accompanying a joint infection. It is steadily progressing.

Case 21.—C. P., a girl, age 7, heavily infected with tuberculosis, displaying primary lesions in tonsils and lung and a hæmatogenous focus in the hip, developed symptoms of miliary tuberculosis and died of tuberculous meningitis. During the course of this fatal illness tubercle bacilli were present in the urine. Though post-mortem evidence is lacking, I think there is no doubt that the kidneys were involved in the miliary process. The absence of pus is probably accounted for by

the small size of the renal lesions. The clinical course and urinary findings parallel several cases of tuberculous meningitis in which post-mortem examination has revealed tubercles in the kidney.

Case 22.—C. M., a boy, age 14, admitted to hospital in January, 1927, without any evident primary focus, but with a hæmatogenous lesion in the ankle, displayed an intermittent pyuria and tuberculous bacilluria. Cystoscopy demonstrated that the tubercle bacilli were renal in origin. No symptoms accompanied these findings. While it is yet too early to know positively the outcome of this case, from his progress it seems likely that he will overcome the renal infection.

Case 23.—N. P., a boy, age 10, admitted to hospital in January, 1927, with an evident primary focus in the lung, and with a hæmatogenous lesion in the knee, displayed a constant pyuria and tuberculous bacilluria, unaccompanied by symptoms. Cystoscopy demonstrated the bilateral renal origin of the tubercle bacilli. This case of renal tuberculosis would unquestionably have been overlooked except for routine examination of the urine. The outcome is uncertain. In my opinion the renal disease may yet heal, though the persistent presence of tubercle bacilli may mean a progressive renal tuberculosis.

Case 24.—W. B., a boy, age 3½, with primary pulmonary tuberculosis and a hæmatogenous lesion in the spine, displayed a constant pyuria and tuberculous bacilluria during the whole period he was under observation. For the greater part of this period he presented no urinary symptoms. During the latter part of his illness he developed frequency and displayed in a mild degree the classic picture of renal tuberculosis. The tubercle bacilli were demonstrated to be bilaterally renal in origin. This case represents a true bilateral renal tuberculosis accompanying a bone lesion. Had amyloid disease not supervened, it is difficult to state what would have been its outcome.

Case 25.—A. C., a boy, age 13, who in infancy passed through an intense tuberculous infection, which clinically resembled miliary tuberculosis, made an apparent recovery. Ten years later symptoms of renal tuberculosis appeared and progressed until his death from uræmia. By cystoscopy it was demonstrated that the tubercle bacilli were bilaterally renal in origin. I would judge that in this case the renal tuberculosis was laid down during his intense tuberculous septicæmia in infancy. It remained latent or quiescent for ten years, and then flared into activity, which progressed until loss of kidney function resulted in death.

Table II.—RESULTS OF ANALYSIS OF URINE IN JUVENILE CASES OF BONE TUBERCULOSIS.

	Urine Negative	Urine Positive
Pott's disease	24	3
Tuberculous hip	19	4
Tuberculous knee	12	1
Tuberculous ankle	2	1
Tuberculous elbow	1	0
	58	9 (13·8 per cent)

JUVENILE CASES WITH SOFT TISSUE TUBERCULOSIS.

For purposes of comparison a similar investigation was carried out upon the urine of a number of patients suffering from tuberculosis in various forms but without involvement of bones and joints. This series is limited to children under 14 years of age. Brief histories of the positive cases follow. Each of the positive cases suffered from widespread tuberculosis. All had

miliary tuberculosis or tuberculous meningitis, and all died long before the report of the guinea-pig inoculation was received. The histories are therefore fragmentary.

Case 26.—R. R., a male infant, age 5 months, heavily infected with tuberculosis arising from a primary focus in the lung, developed innumerable hæmatogenous lesions. The urine contained tubercle bacilli, and post-mortem examination of the kidneys revealed many small tubercles in both of them.

Case 27.—D. C., a girl, age 4, admitted to hospital in April, 1926, with a primary pulmonary focus and evidences of heavy hæmatogenous spread, had tubercle bacilli in the urine. While accurate pathological evidence is lacking, it is probable that the kidneys contained miliary tubercles.

Case 28.—A. S., a girl, age 9 months, with pulmonary tuberculosis and a hæmatogenous infection in the thigh, passed urine containing small amounts of pus and tubercle bacilli. While definite evidence is lacking, it is likely that this also represents a case of miliary tuberculosis of the kidney.

Case 29.—K. M., a girl, age 9 months, when admitted to hospital in December, 1927, was found to be suffering from generalized tuberculosis. She died three weeks later. Sputum collected during the period she was in hospital produced tuberculosis upon inoculation into a guinea-pig. There were no symptoms to attract attention to the genito-urinary tract, but the urine produced tuberculosis upon inoculation into a guinea-pig. It contained no pus. Post-mortem examination revealed a primary focus in the lung with widespread miliary tuberculosis. In addition, the lungs contained patches of tuberculous bronchopneumonia and numbers of miliary tubercles.

Table III.—RESULTS OF ANALYSIS OF URINE IN JUVENILE CASES OF SOFT TISSUE TUBERCULOSIS.

	Urine Negative	Urine Positive
Tuberculous peritonitis	1	0
Miliary tuberculosis	2	2
Tuberculous meningitis	7	2
Pulmonary and mediastinal tuberculosis	9	0
Tuberculous cervical adenitis ..	26	0
	45	4 (8.8 per cent)

DISCUSSION.

This unusually high incidence of positive urines amongst patients with bone tuberculosis (37 per cent in adult and 13.8 per cent in juvenile cases) commands attention. It is far in excess of that generally thought to occur amongst patients with surgical tuberculosis. From conversation with other orthopædic surgeons and from my own experience, it may be stated that 5 to 10 per cent represents approximately the number of adult patients with surgical tuberculosis in whom frank renal lesions develop. Amongst children such renal tuberculosis is extremely rare. During the ten years ending in December, 1926, there were admitted to the Hospital for Sick Children, Toronto, 392 cases of bone and joint tuberculosis, and of these only one (approximately 0.25 per cent) developed frank renal tuberculosis. The most important reason for the discrepancy between this apparent incidence of renal

tuberculosis amongst surgical patients and the much greater incidence recorded in this paper is the manner in which the examination was carried out. Under ordinary circumstances the diagnosis of renal tuberculosis is based upon symptoms. Examination of the urine for tubercle bacilli is not carried out until the patient's symptoms attract attention to the genito-urinary tract. Symptoms of renal tuberculosis (frequency, pain, and urgency) are in reality bladder symptoms, arising from irritation of the bladder, either by implantation of the disease there or by the passage through it of considerable amounts of irritating tuberculous material. It is evident that the disease will usually have existed in the kidney for a considerable period of time and will have reached a moderately advanced stage before vesical involvement occurs. At any rate it is easy, by routine examination of the urine, to demonstrate that many patients with tubercle bacilli in the urine are entirely free from the symptoms of renal tuberculosis, though this lesion certainly exists in some cases and probably does in the remainder. Of this series, 9 (57 per cent) of the adults presented none of the usual symptoms of renal tuberculosis, and 8 (90 per cent) of the children were similarly free from symptoms. This freedom from symptoms is strikingly demonstrated in the juvenile series. Prior to December, 1926, the diagnosis of renal tuberculosis was made only once amongst 392 patients. In this case the diagnosis was based upon symptoms and confirmed by examination of the urine. Since January, 1927, routine examination of the urine, irrespective of symptoms, has revealed amongst 67 patients, 9 who had tuberculous bacilluria and in all probability renal tuberculosis.

In certain of the adult cases the freedom from symptoms is also striking. *Case 11*, whose urine constantly contained tubercle bacilli, and whose kidneys and bladder were found post mortem to be extensively involved, had no urinary symptoms whatever up to his death. *Case 6* was, and is, similarly free from symptoms, though tubercle bacilli are constantly present in the urine. *Case 1* and *Case 13* both passed through a considerable period during which they were free from symptoms, though tubercle bacilli were constantly present in their urine. Later they both developed typical renal symptoms and at the present time are readily recognized as cases of renal tuberculosis. It seems reasonable to believe that all cases of renal tuberculosis develop in this fashion. During the early stages, while the disease is limited to the kidney, they pass through a period during which there are no symptoms to suggest a genito-urinary infection. This symptom-free period may possibly be of great length. Only when bladder involvement has occurred do the typical symptoms display themselves. Patients with symptoms, therefore, are long-standing and relatively advanced cases. Confirmation of this is obtained by examination of such cases in this series. The five adults and the one child who presented typical symptoms were all suffering from well-advanced lesions. The juvenile case (*Case 25*) is particularly interesting. It is probable that his renal lesion was laid down during the period when he suffered from miliary tuberculosis. If this is so, ten years elapsed before the disease had progressed sufficiently to produce symptoms. It is evident, then, that the most important reason for demonstration of so high a percentage of positive urines is the routine examination of the urine of all patients irrespective of the presence

or absence of genito-urinary symptoms. Many patients have tubercle bacilli in the urine and present no symptoms. Patients who have symptoms have relatively advanced renal tuberculosis with bladder involvement.

Two other factors of less importance have contributed to the demonstration of this high percentage of positive urines. The first is the reliance which has been placed upon the guinea-pig test for the determination of the presence of tubercle bacilli. In many laboratories search of the urine for tubercle bacilli is carried out by staining the sediment. In my experience this method will yield positive results only when the organisms are present in considerable numbers. Even in competent hands the time necessary for a proper examination renders it a most uncertain method of making a diagnosis. Practically all the specimens of urine used in this research were examined for tubercle bacilli both microscopically and by guinea-pig inoculation. The unreliability of the former method is evidenced by the fact that the positive results it yielded were only about 20 per cent of those obtained by guinea-pig inoculation. The second factor of some importance has been the persistent re-examination of the urine over long periods of time. Ordinarily, reliance is placed upon one or a few tests of the urine. Rarely, if ever, has such prolonged investigation of the urine been undertaken as has been performed in this research. That this is of importance is evidenced by the number of cases in which the urine was only intermittently positive (*Cases 2, 3, 4, 5, 9, 10, 12, 17, 18, 19, and 22*).

The large number of patients with positive urines revealed by this investigation is therefore the result of: (1) The routine examination of the urine of all patients with surgical tuberculosis irrespective of the presence or absence of genito-urinary symptoms; (2) The use of the guinea-pig test to demonstrate the presence of tubercle bacilli; and (3) The persistent re-examination of the urine over long periods of time. Such a high percentage of positive urines from surgical patients, many free from symptoms, raises important questions as to the significance of the findings. The most important of these queries is concerned with the source of the organisms. Are these tubercle bacilli of renal origin or have they reached the urine from extra-renal foci? In an effort to determine this point the source of the organisms has been investigated, wherever possible, by cystoscopy and ureteral catheterization. For various reasons this operation has not been performed in every case, and in some further cases the urine at the time of examination chanced to be negative. Of the 25 surgical cases with positive urines, tubercle bacilli were recovered from the ureteral specimens of 12. In one other case the renal origin of the organisms was demonstrated by post-mortem examination, and in two further cases the condition of the bladder and ureteral orifices at cystoscopy was such as to make a diagnosis of bilateral renal tuberculosis justifiable, though the ureters could not be catheterized. The renal origin of the organisms was definitely demonstrated therefore in 15 cases. In 6 patients upon whom cystoscopy was performed the ureteral specimens were negative upon guinea-pig inoculation. These were all patients in whose urine tubercle bacilli were present only intermittently, and it seems reasonable to suppose that cystoscopy happened to be performed at a time when organisms were not present in the urine. Upon 4 patients cystoscopy was not

performed. The renal origin of the tubercle bacilli has therefore been demonstrated in more than half of the cases. This is a piece of evidence of particular importance, since several of the adult male patients had lesions in the epididymis or seminal vesicles, which conceivably might explain the presence of tubercle bacilli in their urine. As far as these particular patients are concerned, such contamination of the urine from an extra-renal source is not a factor of great importance. Of the 9 children, only one (*Case 25*) had a genital lesion, and in his case the renal origin of the organisms was demonstrated by ureteral catheterization. Of the 7 adult patients in whom definite information as to the renal origin of the organisms is lacking (upon 4 cystoscopy was not performed, and from the remaining 3 the ureteral specimens were negative), only 3 had demonstrable lesions in the epididymis or seminal vesicles. One may summarize the situation by saying that the renal origin of the organisms has been definitely demonstrated in 15 cases, and of the remainder only 3 had genital lesions from which the urine might possibly be contaminated.

It is probable that the presence of tuberculous lesions in the epididymis does not detract from the accuracy of the assumption that the tubercle bacilli are renal in origin. I am of the opinion, though it has not as yet been capable of proof, that the presence of lesions in the epididymis and seminal vesicles is of itself evidence of the pre-existing presence of tubercle bacilli in the urine. It has been assumed, though without adequate proof, that tuberculosis of the testicle is hæmatogenous in origin. It cannot be denied that when tubercle bacilli are present in the blood-stream they may infect the epididymis, just as they may infect any other organ in the body. Tuberculous epididymitis, however, occurs bilaterally with such great frequency as to suggest that some other factor than hæmatogenous infection is involved. It seems reasonable to believe that a tuberculous epididymitis can arise by retrograde infection from the urethra during the passage of urine containing tubercle bacilli, in a manner similar to that in which gonorrhœal epididymitis arises. Two patients in this series (*Cases 12 and 13*) while under observation with positive urines developed acute lesions in the epididymis. When this occurred there was no evidence to justify the assumption that they were suffering from tuberculous septicæmia. They were improving in general health and were free from fever. I think it reasonable to assume that tuberculous epididymitis in some cases—perhaps in many cases—is an index of the presence of tuberculous bacilluria. I find that this impression is shared by Young,¹¹ who states in his *Practice of Urology*: "In the urinary tract the organ first affected is by far most frequently the kidney, with involvement of the remainder of the tract secondary thereto. . . . Infection of the epididymis takes place in tuberculosis as in gonorrhœa, by means of the cord and not by the blood stream. That cases of hæmatogenous infection occur cannot be denied, but they are few in number."

The renal origin of the tubercle bacilli from most of these cases having been demonstrated, it becomes of importance to determine if possible whether or not they arise from foci of renal tuberculosis. It may be stated at the outset that for most of the patients absolute proof of the presence of renal lesions has not been obtained, nor can it be, until the lapse of time makes

possible the accumulation of sufficient pathological material to permit the actual demonstration of the lesions. It is my impression, however, that the presence of tubercle bacilli in the kidney urine means the presence of tuberculous lesions in the kidney, and in support of this assumption there exists considerable evidence. In the past the occasional demonstration of tuberculous bacilluria in patients free from symptoms has been assumed to be due to the excretion of tubercle bacilli from the blood-stream by the kidney, without its suffering any focal damage in the process. If a true excretory bacilluria can occur, then it must be admitted that tubercle bacilli may possibly reach the urine from the blood-stream without damage to the kidney. There is, however, no sound basis for the belief that the kidney ever excretes organisms into the urine in the manner in which it does the chemical constituents of the blood. The work of Helmholtz and his associates²⁻⁵ clearly demonstrates that the kidney does not excrete organisms in the usual sense of the term; that organisms do not appear in the urine without focal lesions in the kidneys, and that the belief in the power of the kidney to excrete formed particles is based upon errors in technique. They used many varieties of organisms in their experiments, though no tubercle bacilli. There seems to be no basis for the prevalent belief that the kidney can excrete organisms, or indeed any formed particles. In the field of tuberculosis, Medlar^{6,7} has demonstrated that after the intravenous injection of tubercle bacilli into animals, the organisms only appeared in the urine when demonstrable lesions existed in the kidneys. The lesions frequently were microscopic in size and could only be demonstrated by serial section of the kidney, but they were always present when tubercle bacilli were found in the urine. On the basis of this experimental work it may be assumed that true excretion of tubercle bacilli by the kidney never occurs and that the presence of tubercle bacilli in the kidney urine always means the concomitant presence of tuberculous lesions in the kidney.

From the clinical viewpoint there is much evidence which confirms the opinion that tubercle bacilli in the urine means tuberculosis of the kidney. In the positive cases of this series the presence of tubercle bacilli in the urine has practically always been accompanied by pyuria. The quantity of pus has varied greatly; in an effort to measure it roughly it has been customary to record the number of pus cells present in a high-power field of uncentrifuged urine. Centrifugalization of the urine concentrates it to such extremely varying degrees that comparison of the intensity of the pyuria is not easy. In positive urines the number of pus cells has ranged from one to fifty per high-power field of uncentrifuged urine; in none was pus entirely and persistently absent. In general there was a close parallelism between the degree of pyuria, the number of organisms, and the intensity of symptoms when these were present. Pus cells in the urine can only come from inflammatory foci, and are evidence, therefore, that the tubercle bacilli arise in definite foci of tuberculosis, rather than from the blood-stream by the excretory activity of the kidney. Dr. M. A. Shipley's⁹ careful histologic examination of kidneys containing foci of renal tuberculosis has demonstrated that the tubules surrounding the tubercle are often packed with polymorphonuclear leucocytes.

In certain cases the progression of the disease from an early stage free

from symptoms to a later stage with all the classic symptoms also confirms the view that the presence of a tuberculous bacilluria, even though unaccompanied by symptoms, is indicative of renal tuberculosis. Thus *Cases 1 and 13* both passed through a long symptom-free period, during which tubercle bacilli were constantly present in the urine, and later entered into a period in which the tuberculous bacilluria was accompanied by the classic symptoms of renal disease. *Cases 20 and 24* are probably of the same type, though the symptoms are as yet so mild that they would scarcely be recognized were they not searched for. The common symptoms of renal tuberculosis (frequency, pain, urgency) are, after all, bladder symptoms due to extension of the disease to the bladder. It is not unreasonable to feel that tuberculosis of the kidney exists for a long time before bladder involvement, with its accompanying symptoms, develops.

As yet it has not been possible to gather a great deal of post-mortem evidence. Three cases (*Cases 11, 14, and 15*) have come to post-mortem examination, and from one (*Case 16*) an operative specimen has been obtained. These specimens all showed tuberculous involvement. Such findings had been anticipated by the clinical condition of the patient; *Cases 14, 15, and 16* presented all the usual symptoms of tuberculosis before death or operation. While *Case 11* was entirely free from symptoms, the urinary findings were so constant and so marked that no doubt was entertained as to the pathologic lesion which was present. The degree of kidney involvement in *Cases 11 and 14* was not as great as was anticipated. In both, the lesions, though numerous were microscopic in size, and in *Case 11* involvement of the left kidney only was found. As the lesions in both these cases showed well-marked evidences of healing, it seems likely that the primary lesion of the genito-urinary tract occurred in the kidney, with secondary involvement of the bladder, which gave rise to the well-marked symptoms in *Case 14*. The kidney lesions underwent partial healing while the vesical lesions progressed to an extreme degree of tuberculous ulceration.

I am indebted to Professor Klotz for an experimental suggestion which has thrown some confirmatory light upon the problem. From several patients (*Cases 20, 23, 24*) daily specimens of urine were collected for periods of ten days. Each of these daily specimens produced tuberculosis when inoculated into a guinea-pig—in other words, tubercle bacilli were present in the urine every day, though the patients were free from symptoms. Were their presence due to excretion from the blood-stream by the kidney, an equally constant tuberculous septicæmia must have been present. A tuberculous septicæmia so constant and severe could hardly exist without the occurrence of miliary tuberculosis. None of the patients had at that time, nor have they at present, any evidence of miliary tuberculosis. Blood cultures taken during the periods when urine was collected were free from tubercle bacilli. It seems certain, therefore, that the tubercle bacilli came from foci of renal tuberculosis.

If it is granted that the presence of tubercle bacilli in the urine of these patients means tuberculosis of the kidney, it then becomes evident that some of them have reached the stage of apparent cure. *Cases 2, 4, 5, 10, 12, 17, 18, and 22* appear to represent cures. All had tubercle bacilli and pus in the

urine during the early portion of the period of observation and all are now free from these signs and have no symptoms. It would be premature to express too certain an opinion on these apparent cures. The disease may only have become latent, with the prospect that it will spring into renewed activity at a later date. Too little time has elapsed to permit the collection of the pathological material which would give the most definite answer to the question of cure. It must be granted, however, that certain of the cases appear to be cured and some probably are cured.

It is a generally accepted idea that tuberculosis of the kidney is a steadily progressive disease which always goes on to destruction of the kidney. In all probability this opinion is erroneous because based on insufficient evidence. It should be emphasized that the term 'tuberculosis of the kidney' ordinarily is used to describe a well-defined and fairly constant clinical and pathological complex. It is distinguished by definite symptoms (frequency, pain, and urgency), definite urinary findings (pyuria, hæmaturia, and the presence of tubercle bacilli in the urine), and the practically constant finding of a large focus of tuberculosis in one or both kidneys. In other words, as ordinarily used, the term 'tuberculosis of the kidney' applies to advanced lesions of the kidney. It is a matter of clinical experience that such lesions do not heal spontaneously; but all such gross lesions must have passed through a stage in which they developed from the original microscopic tubercles. It is evident that many of the patients reported in this series were in the initial stage of the disease, since they presented none of the symptoms ordinarily associated with the disease, and the urinary findings were slight in degree and sometimes intermittent. If cure takes place it must be of lesions such as these. It is reasonable to suppose that the minute tubercles which represent the primary establishment of the disease in the kidney may undergo one of two fates. They may undergo healing just as do tubercles in other tissues, or they may continue to progress and thus give rise to the large area of tuberculosis which forms the pathological basis of the disease in its full-blown form. It would be extraordinary were the kidney, of all the tissues in the body, the only one incapable of ever overcoming a tuberculous infection.

Medlar⁸ came to a conclusion similar to the above after examination of the kidneys of patients dying of pulmonary tuberculosis. He was able to prove that of 30 patients dying of pulmonary tuberculosis, 22 had renal lesions. None had had symptoms before death. From the histological picture, he is strongly of the opinion that renal tuberculosis can and does go on to healing. The lesions he found were all small; many were microscopic and required serial sections to demonstrate them. Many were surrounded by a zone of fibrous tissue and were in various stages of obliteration by fibrosis. I have received further confirmation of the histologic evidence of healing of renal tuberculosis in a personal communication from Dr. M. A. Shipley.⁹ In examining the kidneys from ten patients with renal tuberculosis of all stages and grades of severity, he has been able to find in all some evidences of attempted healing in the form of fibrosis about the tubercles. In some cases this fibrosis was of high degree, so that cure might be said almost to have occurred. The histologic proof of cure is complicated by the fact that a completely healed tubercle is merely a small fibrous scar with nothing about it

distinctive of tuberculosis. Medlar has demonstrated these scars in more than half of his cases. While there was nothing distinctive about them, their presence in the kidneys of patients suffering from tuberculosis and in kidneys which contain definite tuberculous foci strongly suggests that at least some of them represent healed tubercles. Medlar's conclusions are: (1) Renal tuberculosis is common in advanced pulmonary tuberculosis; (2) Tuberculous lesions of the kidney can and do heal.

It is certain that many patients with pulmonary tuberculosis have lesions in the kidney which, because they are small and because they produce no symptoms, are never recognized unless careful search is made for them post mortem. Not only may they produce no symptoms, but they may also be unaccompanied by any urinary signs, and tubercle bacilli may be absent from the urine. The case whose history follows illustrates this point.

Case 30.—J. T., male, age 36, admitted in March, 1926, with advanced pulmonary tuberculosis and three hæmatogenous foci, presented no symptoms suggestive of renal tuberculosis during the twenty months he was under observation. Careful examination of his urine during the same period demonstrated the absence of pus and tubercle bacilli; yet post-mortem examination of the kidneys revealed many typical tubercles in both kidneys, all showing some evidences of healing. It is evident therefore that renal tuberculosis can exist without symptoms and without tubercle bacilli in the urine. Such a finding is to be anticipated from the pathology of the disease. From its hæmatogenous origin the earliest tubercles are laid down in the cortex. The medulla and pelvis are involved by extension along the urinary tubules. It is quite possible to conceive that at an early stage of the disease none of the tubercles communicate with uriniferous tubules and therefore no tubercle bacilli appear in the urine. The significance of this observation is that one or even more negative urines does not necessarily exclude tuberculosis. Frequently it is only from repeated examinations that any certain conclusions can be drawn. *Case 1* illustrates this point. Even after mild symptoms attracted attention to the urine, it needed a year of repeated examinations to obtain a positive guinea-pig inoculation.

An important point in *Case 30* was the demonstration of tubercle bacilli in the blood-stream on Nov. 11, 1927. That is, there existed a situation such as would permit an excretory bacilluria to occur if such a phenomenon were possible. Yet urine collected on Nov. 17, 1927, contained no tubercle bacilli.

Medlar (*loc. cit.*) made the significant observation that in all the cases of his series in which renal tuberculosis was present, it was present in both kidneys. Because of the hæmatogenous origin of renal tuberculosis both kidneys stand an equal chance of infection, and, ordinarily, both kidneys are infected. Dr. Shipley has made the same observation. In three of six cases in which both kidneys were examined, the renal disease was bilateral. These important observations mean that at the outset practically every case of renal tuberculosis is bilateral. Most of the cases of so-called unilateral tuberculosis must represent an extensive lesion of one kidney and a minor lesion of the remaining kidney. The majority of patients who recover from renal tuberculosis following nephrectomy do so because the tuberculous process which will commonly be present in the remaining kidney goes on to healing. Of the same significance is the observation that the most common cause of late death after nephrectomy for renal tuberculosis is tuberculosis of the remaining kidney. Persson¹⁰ finds that such an occurrence accounts for 44.4 per cent of the late mortality of 24.7 per cent. Young's¹¹ statistics

also show that recurrent genito-urinary tuberculosis is the most common cause of death. *Case 16* illustrates the same point. This patient, with urgent symptoms of renal tuberculosis, was found on cystoscopic examination to have a gross lesion of the left kidney. Six weeks later, when the report of the guinea-pig inoculation was received, tubercle bacilli were found to have been present in the right kidney urine. In the meantime the left kidney had been removed. In spite of the existence of right renal tuberculosis, the patient's symptoms promptly and completely disappeared, and for fifteen months there was no recurrence of them.

The following case strongly suggests the possibility that renal tuberculosis is capable of spontaneous cure :—

Case 31.—A. B., female, age 13, suffering from bovine tuberculosis, presented renal tuberculosis as the only discoverable hæmatogenous lesion. Cystoscopy revealed the lesion to be bilateral. Under rest and heliotherapy she made remarkable progress and became free from symptoms. Though tubercle bacilli are still present in the bladder urine, the left kidney urine is now free from them. It is yet too early to forecast the outcome, but her improvement suggests that for the left kidney at least a spontaneous cure has taken place.

SUMMARY.

It seems evident from the work of Medlar^{6,7,8} and of Shipley,⁹ as well as from the research reported in this paper, that renal tuberculosis is a common occurrence amongst tuberculous patients. Being hæmatogenous in origin it is particularly common amongst patients who have evident hæmatogenous lesions elsewhere—for example, patients with bone and joint tuberculosis. Also because of its hæmatogenous origin, the renal disease tends to be bilateral. There is evidence that at least the early and small lesions have a strong tendency to heal. Healing occurs possibly in the majority of such cases. Less frequently one or several lesions progress and involve a considerable area of the kidney in a caseating tuberculous process. It is this ulceroeavernous process (renal phthisis) which is the pathologic basis of the ordinary case of renal tuberculosis. In its early stages renal tuberculosis is unaccompanied by symptoms. Even the urine may be free from pus and tubercle bacilli (*see Case 30*), though probably this is of infrequent occurrence. The lesion may heal in this early stage so that the patient passes through the whole course of the renal disease without symptoms attracting attention to the genito-urinary tract. Only the urine displays evidence of the disease by the presence of pus and tubercle bacilli. If, instead of healing, the disease progresses, its course is characterized by increasing urinary signs, until finally, with involvement of the bladder, there appear the classic symptoms of renal tuberculosis. It is apparently common for the disease to progress in one kidney and heal in the other.

Acceptance of the fact that tuberculosis of the kidney can heal spontaneously adds to the difficulties of treatment. When it is assumed that renal tuberculosis is always a progressive lesion, and that cure never occurs, it is evident that nephrectomy, when this is possible, offers the only hope of cure. Under such circumstances it is reasonable to advise every patient with tubercle bacilli in the urine from one kidney to have the kidney removed.

Table IV.—SUMMARY OF THE POSITIVE CASES.

This Table summarizes in condensed form all the facts concerning the positive cases. The presence or absence of symptoms and urinary findings is indicated by + and 0 signs. The intensity of symptoms and signs is roughly indicated by the number of + signs; 0+ meaning *intermittently present*, + meaning *slight*, ++ *moderate*, and +++ *intense* degree. The frequency with which tubercle bacilli were found in the urine is indicated by recording the percentage of specimens examined which were positive.

CASE NO.	URINARY SMPTOMS						PERCENTAGE OF SPECIMENS POSITIVE	END-RESULT OF KIDNEY LESION	REMARKS
	Frequency	Urgency	Pain	Pyuria	Hematuria	T.B.			
Adult Cases									
1	+	+	+	++	0	+	100	Still uncertain	General health good, but T.B. constantly present in urine. Symptoms negligible
2	0	0	0	0+	0	0+	12	Apparent cure	In good health and free from symptoms 3 years after T.B. first found in urine
3	0	0	0	0+	0	0+	10	Still uncertain	Health good and urine at present free from T.B., but has recent epididymitis
4	0	0	0	0+	0	0+	14	Apparent cure	Health good, free from symptoms and from T.B. 2½ years after they were first found
5	0	0	0	++	0	0+	7	Apparent cure	Health good, no symptoms 1 year after discovery of T.B.
6	0	0	0	+	0	+	100	Still uncertain	No symptoms, but T.B. constantly present in urine
7	0	0	0	++	0	+	100	Still uncertain	No symptoms, but T.B. constantly present in urine
8	0+	0	0	+	0	+	80	Still uncertain	Health good, symptoms slight, but T.B. constantly present in urine
9	0	0	0	0+	0	0+	25	Still uncertain	Renal disease of recent onset. Failing in health. Heavily infected
10	0	0	0	+	+	0+	25	Apparent cure	In good health and free from symptoms 4 years after T.B. first found
11	0	0	0	+++	+	+	100	Extensive renal involvement	Died of tuberculous meningitis
12	0+	0	0	0+	0+	0+	18	Apparent cure	In good health and free from symptoms 3½ years after T.B. first found
13	+++	++	++	+++	++	+	100	Progressive renal involvement	Dying of renal T.B.
14	+++	++	++	+++	++	+	100	Extensive renal involvement	Died of amyloid disease
15	+++	++	++	+++	+++	+	100	Extensive renal involvement	Died of uræmia
16	++	+	+	++	+	+	100	Definite renal involvement	Temporary recovery after nephrectomy, with recurrence in opposite kidney 15 months later

Continued on next page.

Table IV.—SUMMARY OF THE POSITIVE CASES—continued.

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THE
Table IV.—SUMMARY OF THE POSITIVE

CASE NO	URINARY SYMPTOMS						PERCENTAGE OF SPECIMENS POSITIVE	END-RESULT OF KIDNEY LESION	REMARKS
	Frequency	Urgency	Pain	Pyuria	Hæmaturia	T.B.			
Juvenile Cases									
17	0	0	0	0	0	0 +	16	Apparent cure	Health good and free from symptoms 1 year after T.B. first found
18	0	0	0	+	0	0 +	33	Apparent cure	Health good and free from symptoms 18 months after T.B. first found
19	0	0	0	++	0	0 +	20	Still uncertain	Health good. No symptoms. No T.B. in urine but cystitis persists
20	0 +	0	0	++	+	+	100	Still uncertain	Dying of amyloid disease
21	0	0	0	0	0	0 +	50	Uncertain, P.M. not obtained	Died of tuberculous meningitis
22	0	0	0	0 +	0	0 +	40	Apparent cure	Health good. No symptoms 1 year after T.B. first found
23	0 +	0	0	+	0	+	90	Still uncertain	Health fair. No symptoms, but urine still contains T.B.
24	0 +	0	0	++	0	+	100	Uncertain, P.M. not obtained	Died of amyloid disease
25	+++	+++	+++	+++	+++	0	100	Uncertain; extensive involvement probable	Died of uræmia

tems are present. To admit that many cases of renal disease are present. I think this advice less reasonable. I am sure that large lesions with gross picture. T

whether or not symptoms are present. To admit that many cases of renal tuberculosis heal spontaneously makes this advice less reasonable. I am of the opinion that only small renal lesions heal; that large lesions with gross caseation sooner or later progress to the typical pathological picture. The problem, then, is to distinguish between small and extensive renal lesions. The former may heal spontaneously and should be given an opportunity to do so under conservative treatment. The latter should be treated by nephrectomy if the unilateral distribution of the disease permits this. It is even reasonable to hold that when one kidney is grossly involved and the other involved to a minor degree, the grossly involved kidney should be removed, in the hope that the less involved kidney may recover spontaneously under conservative treatment. The objection may be raised that even though spontaneous cure is possible, to delay surgical treatment is toying with fate. I feel, on the contrary, that these patients should be spared as much kidney tissue as is possible. When the diagnosis of tuberculosis of one kidney is made there is a fair probability that the other kidney is already infected. Even if it is not, nephrectomy leaves the patient with but one kidney to bear the brunt of such future blood-borne showers of tubercle bacilli as may arise from other foci of tuberculosis still active within his body. Renal tuberculosis is not a disease which progresses rapidly. In early cases there is ample time for extended observation before the decision as to operation must be made.

It becomes of importance, therefore, to distinguish between minor and major renal lesions. I believe this can be done upon the basis of symptoms and urinary findings. The minor lesions are unaccompanied by symptoms, and the urinary findings are slight in degree and may be intermittent. Under ordinary circumstances such lesions will be discovered but rarely, and then by chance examination of the urine. But with the arrival of the day when tuberculosis is clearly regarded as a systemic disease will come the time when examination of the urinary tract will be thought as necessary as examination of the chest. Institutions for the care of tuberculous patients will search for the earliest evidences of infection of the urinary tract by repeated routine urinalyses. Under such circumstances minor lesions will be found more frequently, and the problem of their treatment will become of more importance. The major lesions are distinguished by the accompaniment of symptoms (i.e., the presence of bladder involvement); in males, by the presence of lesions in the genitals (epididymis, vas, and seminal vesicles) and by constant urinary findings of more extreme degree.

CONCLUSIONS.

1. By the repeated routine inoculation of urine into guinea-pigs it is possible to demonstrate that tubercle bacilli are present in the urine of a high percentage of patients who suffer from bone and joint tuberculosis (37 per cent of adult patients and 18.8 per cent of juvenile cases).
2. In many cases this tuberculous bacilluria is unaccompanied by the ordinary symptoms of renal tuberculosis.
3. There is no evidence to support the belief that the presence of tubercle bacilli in the urine is the result of an excretion of them from the blood-stream by the kidney.
4. It is reasonably certain that the presence of tubercle bacilli in the kidney urine means the presence of tuberculous lesions in the kidney.
5. Tuberculous lesions of the male genitals probably arise secondarily to tuberculous lesions in the kidney and the presence of a tuberculous bacilluria.
6. The initial renal lesions frequently heal. Less frequently they steadily progress to complete destruction of the kidney and death of the patient.
7. Minor and major lesions probably can be distinguished from each other on a basis of symptoms and urinary findings.
8. Minor lesions should be treated conservatively because of their tendency to heal; major lesions should be treated surgically where this is feasible.
9. In every case of tuberculosis, but in particular in those which present evident lesions of hæmatogenous origin (c.g., bone and joint tuberculosis), the possibility of renal infection should be borne in mind. Examination of the urine and its inoculation into guinea-pigs should be made routine, instead of being delayed until symptoms appear. Careful surveillance of the urinary tract is as necessary as repeated examination of the chest, and this can best be carried out by repeated routine examination of the urine.

It is a pleasant duty to acknowledge my indebtedness to many associates for their co-operation in the multitudinous details which have made this research possible. In particular my thanks are due to Professor Oscar Klotz and Dr. M. A. Shipley, of the Department of Pathology of the University of Toronto, for examination of the pathological specimens; to Dr. G. W. Loughheed and Dr. A. C. Norwich, of the Pathological Department of Christie Street Hospital, D.S.C.R., for many urinalyses; to Dr. I. H. Erb and Dr. Eileen Boyd, of the Pathological Department of the Hospital for Sick Children, Toronto, for many urinalyses; to Dr. R. M. Price for the supervision of many guinea-pig tests and blood cultures; and to several generations of interns for their assistance with the clinical material.

REFERENCES.

- ¹ PEABODY, C. W., *Ann. of Surg.*, lxxv, 95.
- ² HELMHOLTZ, H. F., and MILLIKIN, F., *Amer. Jour. Dis. Child.*, xxix, 497.
- ³ HELMHOLTZ, H. F., and FIELD, R. S., *Ibid.*, 506.
- ⁴ HELMHOLTZ, H. F., and FIELD, R. S., *Ibid.*, 641.
- ⁵ HELMHOLTZ, H. F., and FIELD, R. S., *Ibid.*, 645.
- ⁶ MEDLAR, E. M., and SASANO, K. T., *Amer. Rev. Tuberc.*, x, 370.
- ⁷ MEDLAR, E. M., *Wis. Med. Jour.*, xxv, 59.
- ⁸ MEDLAR, E. M., *Amer. Jour. Pathol.*, ii, 401.
- ⁹ SHIPLEY, M. A., Personal communication concerning research now in progress.
- ¹⁰ PERSSON, M., *Ann. of Surg.*, lxxxi, 526.
- ¹¹ YOUNG, *Practice of Urology*, 1926, i, 278-280, 324.

SPECIAL ARTICLES
ON SURGICAL TECHNIQUE.

OPERATIONS FOR MALIGNANT DISEASE OF THE PHARYNX.

BY WILFRED TROTTER,

SURGEON TO UNIVERSITY COLLEGE HOSPITAL, LONDON.

To separate the operative surgery of a region from its general surgery is not perhaps so natural and convenient nowadays as it was formerly and at a time when anatomical practicability was not only the primary but also the predominant characteristic of a satisfactory operation. At that time many operations were designed so as to have for their object the removal of a definite part or organ. In these circumstances the surgeon could justifiably describe as perfectly well defined and formal procedures such operations as excision of half the tongue, of the upper jaw, and of the larynx for malignant disease. Thus it was possible for operative surgery to exist as a quasi-independent art having no very close relation to pathological requirements.

To-day, however, we are no longer restricted to these simplified and so to say diagrammatic procedures, and thereby much ineffective treatment and useless mutilation are avoided. The modern operation for cancer is primarily designed to remove a disease rather than to remove an organ. It is no longer evolved, as it were, in an anatomical vacuum, but in close relation with an environment of pathological and clinical fact. These considerations apply especially to the surgery of the nasal cavity, the mouth, the pharynx, and the larynx. Operations for malignant disease in these parts depend for success on full familiarity with the pathological conditions and on very exact diagnosis of the individual case, quite as much as they do on operative technique.

In the parts of the pharynx with which we are here concerned the only form of malignant disease that is found, with the rarest exceptions, is the squamous-celled epithelioma. This tumour shows even in this small area wide variations in its amenability to treatment, and any operation must be planned in relation to these. To estimate the task before him in a given case the surgeon must know the exact point of origin of the growth, the clinical type it conforms to, and the extent to which it has spread. No method of examination can at present provide these data infallibly, or can yet take the place of a judgement trained by the patient study of cases.

The region of the pharynx we are to consider corresponds roughly with that described by anatomists as the laryngeal part, and extends from the tip

of the epiglottis to the lower border of the cricoid cartilage. Epithelioma here permits of classification into four well defined groups, for each of which a special type of operation is necessary.

1. *Superior Group*.—Growths of the epiglottis or of the glosso-epiglottic fossa.
 2. *Lateral Group*.—Growths primary in one of three situations: (a) the aryepiglottic fold, (b) the pyriform sinus, (c) the lateral pharyngeal wall.
 3. *Posterior Group*.—Growths of the posterior pharyngeal wall.
 4. *Inferior Group*.—Growths of the post-ericoid pharynx.
- From the technical point of view the last two groups are definitely marked off from the first two. In *Groups 3 and 4* the affected part of the pharynx, if removed, cannot be reconstituted by any natural process of healing without serious disability, so that a successful operation must necessarily include a definite plastic procedure to replace the excised part. These two groups therefore naturally fall into a distinct chapter of the surgery of the pharynx, and may conveniently be left out of consideration here.

OPERATIONS FOR GROWTHS OF THE SUPERIOR AND LATERAL GROUPS.

GENERAL CONSIDERATIONS.

In undertaking an operation for epithelioma of the pharynx the surgeon's attention must be given to three concurrent themes: (1) The exposure and removal of the growth; (2) The prevention or limitation of sepsis; (3) The preservation of function. They are of equal importance to substantial success, and judgement is necessary to give each its proper weight without prejudice to the others.

Exposure and Removal of the Growth.—Exposure of the growth is a purely anatomical matter and will be referred to in the description of each operation.

Removal of the growth can be dealt with in more general terms. It is not possible to define precisely what can be considered an adequate margin of healthy tissue about the tumour. There is no point in which correct practice is more dependent on experience. In general it may be said that hard growths of the superficial or the fungating type are adequately dealt with by excising them with quite narrow margins of healthy tissue. In the excision no structure should be spared on the one hand, and on the other nothing should be removed that does not come within the prescribed distance from the growth. I prefer to use the scalpel rather than the diathermy knife, because it allows of primary suture of the wound and a finer apposition of one's nearness to the indurated region.

The Lymphatic Gland Dissection.—The view that a bilateral complete gland dissection should form part of the treatment of any epithelioma of the pharynx is not confirmed by an extended experience. In laterally placed growths a dissection of one side of the neck gives excellent results and can generally be combined with the pharyngotomy. In special cases where there

is reason to reduce the treatment to the simplest possible procedure, the gland operation may be omitted if the situation is clearly explained to the patient and he can be kept under periodical observation. In dealing with growths of the glosso-epiglottic region the gland dissection cannot be combined with the pharyngotomy: this must precede or follow, and it should usually be bilateral.

The Control of Infection.—Sepsis is the chief cause of death after operations on the pharynx. It takes two forms—infection of the lungs, and infection of the neck. The best prophylactic against all forms of sepsis is an edentulous mouth with soundly healed gums. The cleansing influence of removing the teeth is best seen in its effect on a foul ulcerating growth, where it is sometimes astonishing. Unfortunately the full effect takes a long time to appear, and it is not usually wise to wait longer than three weeks after the teeth have been extracted. Since a recent clearance of the mouth—although it probably always diminishes the risk of a pharyngotomy—can give no complete assurance of greatly reduced sepsis, it is not perhaps justifiable always to insist rigidly on its being carried out as a preliminary. When the surviving teeth are many and healthy, or there is some other special reason against adding the burden of a large clearance to what the patient already has to bear, a pharyngotomy may justifiably be undertaken without it, but should then be reduced to the simplest terms possible.

Infection of the lungs, when it occurs, seems practically always to be set up during the actual operation. The best precautions against it are preliminary tracheotomy with incessant attention to water-tight packing of the airway during the operation, light chloroform anaesthesia aided by cocaine packs whenever possible, and gentle, bloodless, and reasonably quick operating. The control of infection of the neck should be constantly in the surgeon's mind throughout the operation. At the end of the gland dissection and before the pharynx is opened, the sternomastoid should be firmly stitched over the carotid vessels to the prevertebral muscles. In this way primary union of the most dangerous part of the neck wound can usually be attained. While the pharynx is open all raw surfaces should be carefully protected from contact with pharyngeal mucus. After the growth has been removed every attempt should be made to secure watertight closure of the pharynx. If this is impossible, the margins of the mucous membrane should be stitched to the skin all round the opening so as to leave no raw surfaces. After a few weeks the fistula that results, now much reduced in size, can easily be closed under local anaesthesia. If the pharynx has been successfully closed, the neck wound should be left unsutured and filled with sterilized boric acid powder. Something very little short of primary union is obtained in this way.

The most serious danger of uncontrolled infection of the neck is secondary hæmorrhage. It is particularly insidious and may be delayed till no more than an innocent-looking sinus is left. The foregoing general precautions against sepsis are the best safeguards against secondary hæmorrhage, but one special and not very obvious one should be mentioned. When, in the preliminary stage of the operation, any branch of the carotid is divided, as for example the superior thyroid invariably has to be, it should be ligatured as far as may be from the parent trunk. In this way is left a blind end

perhaps an inch long in which firm clotting can occur, whereas if the ligature is applied close to the carotid, a very small slough can liberate blood from the main artery itself.

The Preservation and Restoration of Function.—It is characteristic of operations for growths of the upper and lateral groups that, in cases at all favourable, no plastic measures in any way elaborate are necessary. Removal of the epiglottis down to the vocal cords together with a large part of the base of the tongue abolishes swallowing for a time, and the patient usually has to be fed through a tube for two or three weeks. In due course, however, swallowing becomes completely normal.

In the lateral group, removal of the edge of the epiglottis, the aryepiglottic fold, and the arytenoid cartilage permits of complete recovery of function with the exception of some huskiness of the voice. When much of the cricoid cartilage has to be taken away, and the party wall between pharynx and larynx is thus destroyed, the normal separation has to be restored by building up a new party wall out of double skin-flaps. In such extensive cases, however, the prospects of cure by excision are small, and it is better, when the exposure stage of the operation is complete, to deal with the actual growth by implantation of radium, the pharynx wound being left open and the mucous membrane all round stitched to the skin. Early growths on the aryepiglottic fold, in the pyriform sinus, and on the lateral wall are usually curable with no disability. The most favourable of all is the not uncommon localized tumour on the lateral wall; this should give results as favourable as does the cutaneous type of epithelioma of the lip.

TECHNIQUE OF OPERATIONS.

Tracheotomy.—An opening in the windpipe is necessary during the operation, and for a week or ten days after on account of œdema. For the latter reason laryngotomy is inadmissible, and tracheotomy is necessary. Two points only in the technique of this need be mentioned. Whenever the isthmus of the thyroid is seen a segment of it should be excised: and the trachea should be opened not by the traditional vertical cut but by the removal of a disc $\frac{1}{4}$ to $\frac{1}{3}$ in. in diameter. These measures in no way hinder the healing of the wound, and they make it possible to change or replace the tube at any time with perfect ease and safety. StClair Thomson's modification of Durham's tube is indispensable. It is a fact that does not seem widely known that in middle-aged patients when the windpipe is opened the breathing often, and indeed usually, becomes at once periodic in type. Apparently the loss of the stimuli due to the passage of air through the larynx causes the mechanism of respiration to change to a more chemical type in which it depends directly on the state of the blood. The long periods of apnoea that now occur seem to be quite harmless, but are apt to cause alarm to the inexperienced.

OPERATION FOR GROWTHS OF THE SUPERIOR GROUP.

Median (Anterior) Translingual Pharyngotomy.—Growths of this group, as they tend to invade the tongue, cannot be exposed properly by lateral

pharyngotomy without lateral division of the mandible, which, in comparison with the method here advised, is a clumsy and unjustifiable complication.

The advantages of the median operation are that when exactly carried out it is almost entirely bloodless, that it follows a natural line of cleavage, that infective complications are almost non-existent, and that it gives the best access in the direction in which it is most needed.

Incision.—After preliminary tracheotomy and plugging of the pharynx an exactly median incision is made over the lower lip, the chin, and the submental region to the top of the thyroid cartilage.

Exposure of the Growth.—The cut is deepened first below, until the mylohyoid muscle has been divided, and then the interval between the geniohyoid muscles is found close to the hyoid bone. This interval is the best guide to the middle line, and it is opened up as far as the jaw. The incision is now carried through the lip down to the mandible, which is sawn through exactly in the middle line. The same line of cleavage is followed through the floor of the mouth to the tongue, which is then split in the same way. The two halves of the mandible separate readily and the splitting of the tongue is carried backwards. In this procedure very little more than the mucous membrane needs cutting, the tissues for the most part opening symmetrically under gentle blunt dissection. (*Fig. 38^o*)*

Removal of the Tumour.—When the median incision comes within range of the growth, the excision is begun by cutting outwards laterally at the proper distance. As soon as the growth has been separated from the tongue it can be drawn upwards into the wound, the aryepiglottic folds divided, and a horizontal cut made across the epiglottis just above the vocal cords. The growth is now free from everything but the body of the hyoid bone, and all that remains is to disarticulate this latter from the great cornu on each side. Neither of the lingual arteries is as a rule seen or divided.

Closure of the Wound.—The large gap between the larynx and the tongue made by the removal of the tumour is closed by large sutures passed deeply through the tissues on each side. The absence of the body of the hyoid permits the parts to come together easily. The median incision in the tongue is stitched along the dorsum, over the tip and on the under free surface. Wiring the jaw is not necessary. It is fixed by strong catgut stitches passed through the tissues close to the bone. The lip is stitched on both surfaces and then the skin over the chin is closed. The submental part of the skin incision is left open for drainage.

After-treatment.—This is carried out on the lines laid down for the

* NOTE ON ILLUSTRATIONS.—Although tumours of the pharynx permit of grouping into types according to their situation, and actual cases conform to these with considerable exactitude, every case is in its finer details unique and presents an individual technical problem. To illustrate usefully, therefore, the final stage of the operation—that is, the actual removal of the tumour—would involve the representation of a great amount of detail. On this account the illustrations given here are intended merely to show the broad anatomical outlines governing the methods of approach. These anatomical studies I owe to Dr. H. A. Harris, who has expended time and thought on them with unlimited generosity. The dissections were made by Miss A. E. Russell, Demonstrator in the Institute of Anatomy, University College, to whose skilful help I am greatly indebted.

The incisions used have not been limited to those of the operations on the living subject, as it was thought a somewhat wider exposure might be more instructive.

lateral operation (p. 495), but is much simpler in every way. Little is gained by allowing the patient to attempt natural swallowing for ten days or a fortnight. After this he should be encouraged to try as soon as he feels inclined. Normal swallowing may be expected ultimately with complete confidence.

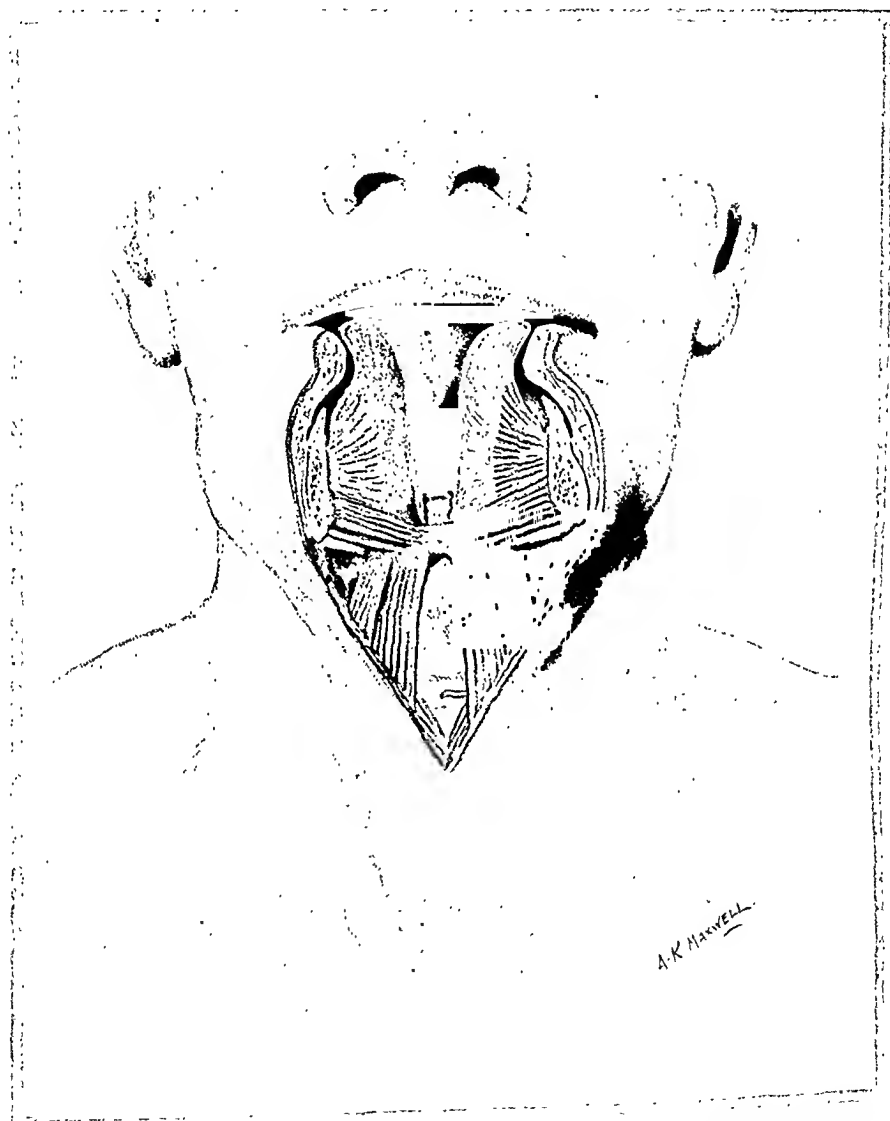


FIG. 389.—Anterior translingual pharyngotomy, exposure stage completed. The epiglottis has come into view, and a growth in that region can now be defined and removed.

The Gland Operation.—There is no question of the gland operation and that for the primary growth being done at the same sitting. It is therefore an advantage of the method that the regions involved in the two procedures

are entirely distinct. As a general rule the extent and gravity of the primary growth in these cases are uncertain, and it is therefore often prudent to postpone the gland operation until it is known whether the prospects of cure justify the rather formidable addition of a bilateral dissection of the neck.

OPERATION FOR GROWTHS OF THE LATERAL GROUP.

Lateral Transthyroid Pharyngotomy.—This operation is based on two anatomical facts: (1) That the pharynx is shielded from access laterally by



FIG. 390.—Lateral radiogram of the neck. To show the relation of the pharynx to the laryngeal skeleton and the surface. All the parts shown appear in the original photograph except the superior cornu of the thyroid, which has been added. The relation of the epiglottis to the hyoid bone is well seen, and it is plain that lateral access to the laryngeal part of the pharynx can be obtained only if the thyroid ala and great cornu are removed.

the laryngeal skeleton, namely the thyroid ala, the great cornu of the hyoid bone, and the thyrohyoid ligament connecting them posteriorly; and (2) That these structures can be removed, leaving the wall of the pharynx and any growth within it undisturbed and unopened. (Fig. 390.) Thus it is possible

at this stage when dealing with early growths to palpate the tumour through the pharyngeal wall, to estimate its extent and situation, and to choose the best place for the incision into the pharynx.

Incision.—The preliminary tracheotomy having been done, an incision is made downwards from a point just behind the angle of the jaw for 4 or 5 in. according to the length of the neck. It is more nearly vertical than the sternomastoid, to which it is therefore oblique. In the cases where it is not intended to attempt a gland dissection the incision may be placed an inch further forwards, starting from below the mandible and extending vertically downwards. (*Fig. 391.*)

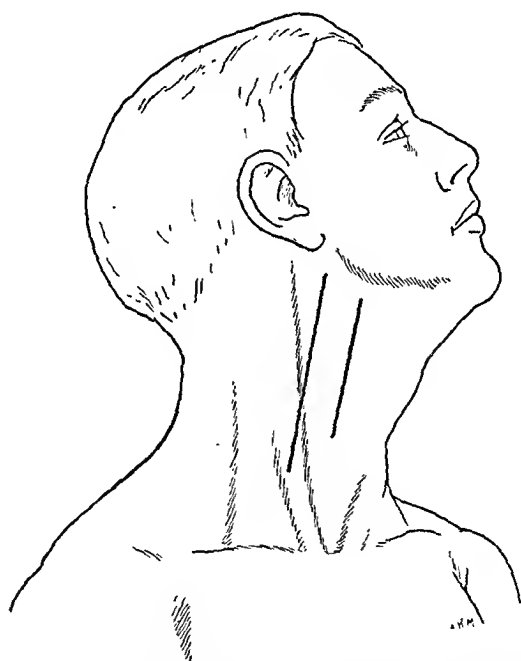


FIG. 391.—Incisions for lateral transthyroid pharyngotomy. The posterior and longer incision is used when a gland dissection is also done. The anterior incision is used when pharyngotomy alone is intended.

The Gland Dissection (Fig. 392).—The anterior edge of the sternomastoid is defined and the muscle freely retracted backwards. To make this easier a transverse cut may be made into the muscle at the lower end of the wound. The usual gland dissection is now done, the parts removed including the internal jugular vein, but not as a rule the submaxillary gland. The superior thyroid artery is ligatured well away from the carotid. The anterior edge of the sternomastoid is now brought over the carotid vessels and sutured with catgut to the prevertebral muscles behind the pharynx. In this way the cavity of the gland dissection is shut off. It may be drained through a puncture behind the sternomastoid. If the seclusion of the carotids seems imperfect above, as it often does, the digastric tendon is divided and the posterior belly of the muscle turned back to fill the gap.

Exposure of the Pharynx.—A vertical incision is now made downwards over the lateral aspect of the larynx midway between its anterior and posterior borders. This divides the infrahyoid muscles down to the cartilage,

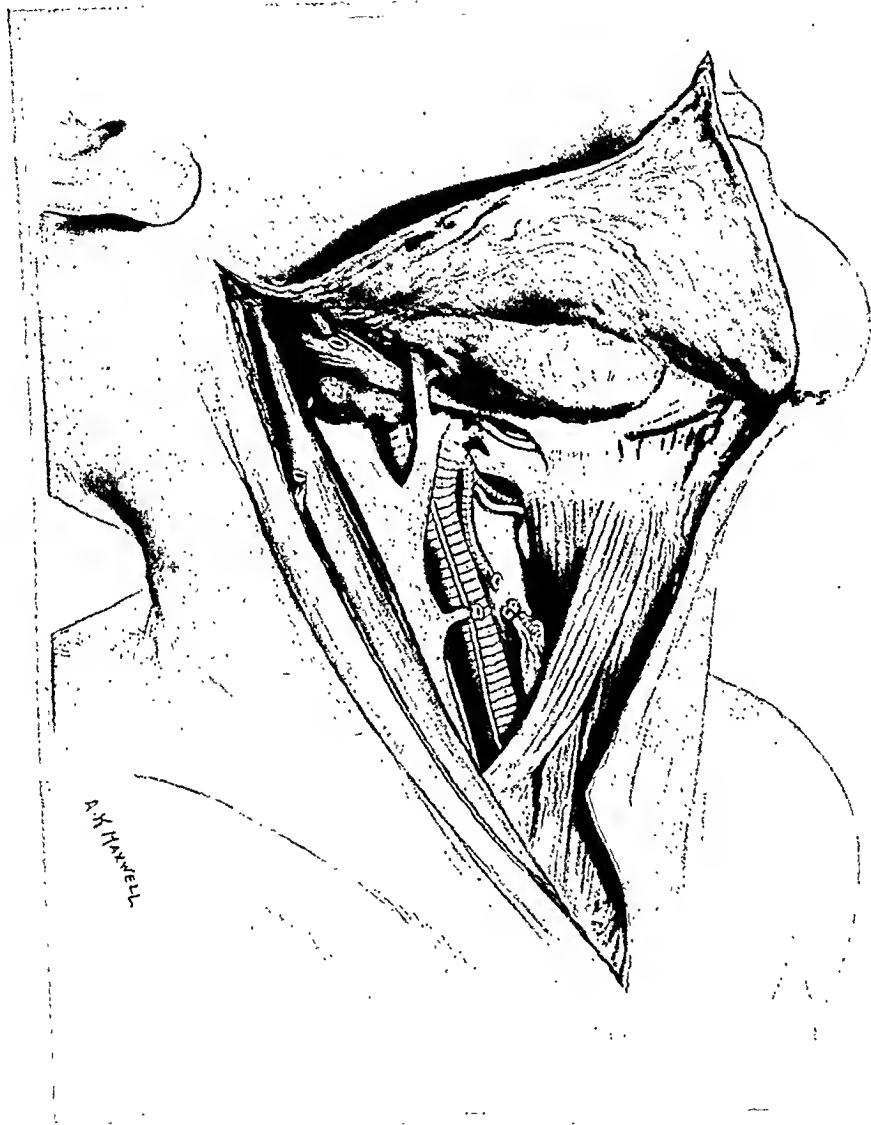


FIG. 392.—Lateral pharyngotomy, early stage. The superior thyroid artery and vein have been divided and the carotid vessels exposed. The sternomastoid will now be retracted further, the common facial vein divided, the internal jugular vein ligatured above and below, and the gland dissection completed.

off which they are reflected forwards and backwards, exposing the thyroid ala and the great cornu of the hyoid. At this stage the superior laryngeal vessels and nerve are divided. In reflecting the muscles backwards care is

taken to keep them in a continuous sheet and to include the inferior constrictor. This flap is useful later to cover in the suture line in the pharynx. The great cornu and the thyroid ala are now separated from the underlying

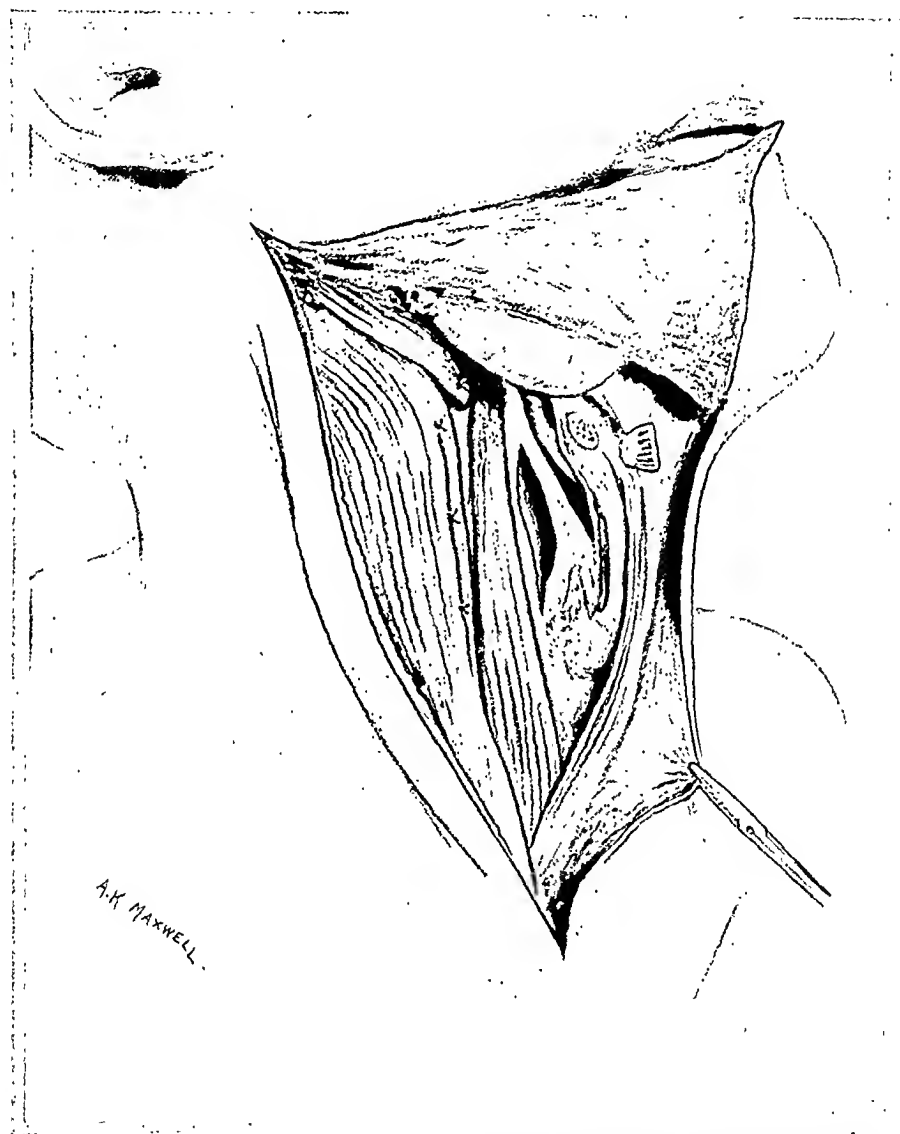


FIG. 393.—Lateral pharyngotomy, exposure stage completed. The gland dissection has been done, and the resulting cavity secluded by suture of the sternomastoid and digastric posterior belly to the provertebral muscles. The infra-hyoid muscles have been divided vertically and reflected forwards and backwards. The thyroid ala and great cornu have been removed. In the exposed pharyngeal wall two illustrative incisions have been made. The anterior one, through which the epiglottis can be seen, might serve to expose a growth of the aryepiglottic fold. The posterior one is such as might begin the excision of a growth of the lateral wall.

pharynx wall. It is easy to do this without opening the latter. Circumspection is necessary during this step lest the growth should have penetrated the

pharynx and be involving the cartilage. This is a serious complication and involves removing the ala in continuity with the growth. In all early cases the ala is quite free from the underlying tissues. It is now divided vertically at the junction of its anterior and middle thirds, and is removed with the thyrohyoid ligament and the great cornu. The lateral aspect of the pharynx is now freely exposed and through its lax wall the tumour can be clearly felt. (Fig. 393.)

Removal of the Tumour.—The raw surfaces of the wound are protected with gauze soaked in an antiseptic, and the pharynx is incised. The site of the incision is determined by that of the tumour. If, for example, the tumour is in the lateral wall, the incision that opens the pharynx will serve as part of that by which the excision is effected; if again the tumour is on the ary-epiglottic fold, a vertical incision must first be made to expose it. As soon as the lumen of the pharynx is reached the glottis is carefully plugged with a strip of gauze soaked in 10 per cent cocaine solution. Everything is now ready for the excision of the growth, which can be done deliberately under guidance of the eye and finger. It is desirable to sear the surface of the ulcer with the cautery before the removal is begun.

Closure of the Pharynx.—There are three ways in which the pharynx may be dealt with, according to the size of the tumour: (1) Immediate closure; (2) Suture of pharyngeal mucosa to the skin so as to leave a large fistula and cover in all raw surfaces; (3) Reconstitution of the septum between larynx and pharynx by skin-flaps. The third course, for reasons already given, will not be dealt with here.

Immediate closure is the normal course. When a small growth of the aryepiglottic fold has been excised, the wound thus made locally can most often be sutured separately. The incision in the pharynx wall is then stitched and reinforced by bringing together the muscular flaps that have been turned off it. If a tumour of the lateral wall has been excised, the gap can usually be closed and covered with the muscle-flaps. The neck wound is then filled with sterilized boric acid powder and left unsutured. In favourable cases no leakage from the pharynx occurs and then the neck wound heals quickly; as it does not gape widely the absence of stitching causes very little delay. In such circumstances healing may be complete in three weeks. If leakage from the pharynx can be postponed even only for three or four days, there is little risk of serious infection.

When the pharynx cannot be closed the best course is to suture its wall all round the opening to the skin of the neck. In this way all raw surfaces are covered in, and there is usually but little infection. In three or four weeks a very simple procedure under local anaesthesia suffices to close the now greatly contracted fistula.

After-treatment.—In all cases a No. 12 rubber catheter is passed into the oesophagus through the mouth and left in for three or four days. After that it is passed for individual feeds. The tracheotomy tube is necessary for about a week. The time when it can be taken out is determined by plugging its opening for an hour or two. The neck wound is dressed frequently and carefully watched in case any local collection of pus should form. A normal pulse is the best evidence that no serious infection has occurred.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD.

PROFESSOR LERICHE AT STRASBURG.

IN 1913 Professor René Leriche called the attention of surgeons to the operation of peri-arterial sympathectomy as a method of permanently improving the nutrition and circulation of a limb, this being necessary especially, say, in the case of intractable ulcers. Incidentally the procedure was found to relieve pain, and was quickly and widely adopted for cases with loss of good circulation and pain in an extremity. In 1915 Leriche adopted the operation for the cure of causalgia after war wounds. Since then it has been widely used. As was to be expected, the results of such an operation, becoming so widely employed before the physiological and pathological *raisons d'être* were correctly understood, have been most varied. The position of peri-arterial sympathectomy cannot be better summed up than in the words of Leriche himself, in a paper in the *Annals of Surgery* (September, 1928) in which he says: "The surgery of the sympathetic system meets two kinds of difficulties, those which spring from our physiologic ignorance, those which spring from our pathologic ignorance. On one side we do not know the exact significance of the branches that we cut, on the other side we are ignorant, as a rule, of the cause and the exact mechanism of the diseases we wish to cure."

With such confused ideas of peri-arterial sympathectomy prevalent it was only natural that the visit of a party of British surgeons to the Clinic of Professor René Leriche at Strasburg should be arranged, and an attempt made to obtain so to speak 'a pure culture' from the great exponent of the surgery of the sympathetic system. They were not disappointed. In order to appreciate Professor René Leriche and his work one cannot do better than quote him again. In his inaugural lecture as Professor of Clinical Surgery of the University of Strasburg delivered on March 13, 1925, he says in his opening remarks, "Vous ne me connaissez pas. J'avais besoin de prendre contact avec vous autrement qu'en paroles. J'ai préféré que vous me jugiez d'abord aux actes."

To appreciate the personality of the man is to appreciate more fully the quality of his work. He is not a mere tyro applying a universal remedy for every and any type of case. Each case is fully studied, and a real attempt is made to discover and estimate the underlying physiological and pathological processes and their relative importance.

The operative technique employed is of the simplest. Moreover, it would

be impossible to see a demonstration of more deft and less injurious dissection on the living subject than that seen in Professor Leriche's operating theatre, under local, spinal, or general anæsthesia. His work illustrates very aptly the ideal and trend of modern surgery—that the surgeon is no longer content to be the mere technician; that is a primary essential, but in addition he must be able to blend with his technique the clinical findings, the pathological processes, and consequent physiological errors, before attempting their correction. An ideal perhaps which has been best realized and attained by the neurological surgeon or—if the term be preferred—surgical neurologist.



FIG. 394.—Professor Leriche in his Clinic.

Two very full days were spent in the Clinie with Professor René Leriche and his associates. The spirit of camaraderie amongst professor, associates, nurses, and patients has to be seen to be appreciated, and an atmosphere of enthusiasm pervades the whole Clinie.

Operation 1. CANCER OF THE BREAST in a woman, age 27. This was the first operation witnessed. Iodine was used for the skin, open ether was the anæsthetic. A 'Halstead' operation was employed. The light was artificial, and the towels, operating gowns, etc., were blue. Fifteen artery forceps only were employed and there was practically no loss of blood. The operation time was one hour.

2. SYMPTOMS INDICATIVE OF A RIGHT-SIDED CERVICAL RIB in a woman,

age 26. The X rays showed what was called a prominent transverse process of the 7th cervical vertebra. The anæsthetic was local $\frac{1}{2}$ per cent novocain and adrenalin. The incision was oblique, parallel to the border of the sternomastoid. Every structure in the operation field was quickly, deftly, and delicately exposed and demonstrated. No cervical rib was found. The scalenus anticus was divided at its insertion and a peri-arterial sympathectomy of the subclavian artery was performed. The operation time was 50 minutes.

3. VARICOSE ULCER.—A man, age 45, had had a large varicose ulcer on the left leg for ten years. The internal saphenous vein had been tied and a portion removed five years ago; the ulcer had healed after this operation but had broken down again when he had re-commenced his work. The anæsthetic was intraspinal. A peri-arterial sympathectomy of the superficial femoral artery was done, followed by a wide excision of the ulcer, including all the discoloured skin around it. In ten days' time the raw surface would be skin-grafted. The result seemed a foregone conclusion.

Many such cases of healed chronic ulcer were shown in the intervals between the operations. These patients had previously had ulcerated legs for many years, up to twenty. They had spent months of their lives in hospital. They were all enthusiastic in praise of the operative treatment of Professor Leriche. They were all working and many of them doing heavy labouring work.

Cases of Volkmann's ischæmic contracture treated by peri-arterial sympathectomy were shown, one in particular in which there had been no demonstrable lesion of the bone, only a wound involving the soft parts of the forearm with no obvious injury to the nerves. A typical contracture had developed which had completely disappeared forty days after peri-arterial sympathectomy. Many cases of diseases in bones and joints which had been treated by peri-arterial sympathectomy and lumbar ramisection were also shown.

Professor Leriche discussed "the problem of osteo-articular diseases of vasomotor origin" in a paper published by him in the *Journal of Bone and Joint Surgery* for July, 1928. He used the cases shown to demonstrate to the surgeons present his thesis of the sequence of events, namely, trauma, hyperæmia, hydrarthrosis, and osteoporosis. If the latter affects the subchondral portion of the bone, then the cartilage becomes detached and traumatic arthritis develops.

Case 1. A man, age 48, treated for nine months after an injury for tuberculous disease of the wrist-joint by immobilization. Improvement commenced immediately after brachial peri-arterial sympathectomy and progressed to complete recovery.

2. A man who had had a severe injury to the left foot early in 1926 had subsequently developed pain for which no treatment was of any avail. Wasting of the calf, thigh, and buttock followed and he had been in bed for months, being looked upon as a malingerer. Radiography showed extensive osteoporosis. Lumbar ramisection (November, 1926) cured the pain.

He had completely recovered, and the osteoporosis had now disappeared. This is evidently the same case as that mentioned in the *Journal of Bone and Joint Surgery* (July, 1928).

3 and 4. Both these had been diagnosed as cases of arthritis, probably tuberculous, following accidents to the wrist. Cure and reconstitution of the bone had followed brachial sympathectomy.

5. Similar to the above, but in the ankle-joint, following an incomplete fracture of the internal malleolus.

In every case complete sets of radiograms were shown.

On the second day the following operations were witnessed :—

Operation 1. PLATING OF A FEMUR, seven days after the fracture, in a man, age 50. Spinal anaesthesia was employed; mechanical traction was maintained during the operation on a metal pin passed through the leg above the os calcis and anterior to the tendo Achillis.

The incision was made on the external aspect of the thigh, directly through the muscles, to expose the site of fracture. A six-screw Lane's plate was employed. The operation time was one hour.

2. *BRACHIAL ARTERIECTOMY* in the case of a man, age 50, who had suddenly developed pain in the forearm and hand some days previously, with evident failure of the circulation, due to embolism or thrombosis in the brachial artery just above the bifurcation into radial and ulnar branches. There was no demonstrable cause to be found for the embolus.

Professor Leriche expounded the views advanced by him at the thirty-first French Congress of Surgery, Strasburg, 1921, that obliteration of an artery should be treated by arteriectomy so that the collateral circulation might be more surely secured; the irritation of the peri-arterial sympathetic at the level of the lesion is thus surely removed.

The lesion in this man was demonstrated, and the lower portion of the brachial and upper portion of both the radial and ulnar arteries were removed. The operation time was 65 minutes, the anaesthetic being open ether.

3. *PERI-ARTERIAL SYMPATHECTOMY OF THE INFERIOR MESENTERIC ARTERY* for the relief of pain in a case of inoperable cancer of the rectum. The patient was a man, age 24. He had already had a colostomy done for relief of obstruction due to cancer of the rectum. The pain caused by the cancer had become unbearable, and by a proper excision of the sympathetic plexus from the front of the aorta and around the inferior mesenteric artery, Professor Leriche was certain that the pain could be relieved.

The patient was anaesthetized with open ether, placed in a high Trendelenburg position, the intestines were packed off, and the sympathetic trunks on the anterior aspect of the aorta identified and divided low down. They were then followed up and removed as far as the inferior mesenteric artery.

Cases illustrating the use of sympathectomy in the relief of pain and traumatic oedema were also shown. Professor Leriche then explained the operation for removal of the inferior cervical sympathetic ganglion as practised by him. The details were the same as those he published in

Lyon Chirurgical, November–December, 1926. He showed cases in which this operation had been successful in asthma, paroxysmal tachycardia, and angina pectoris.*

OBSERVATIONS ON JUVENILE OBLITERATING ARTERITIS: RESULTS OF TREATMENT BY ARTERIECTOMY AND EPINEPHRECTOMY.

BY PROFESSOR R. LERICHE AND DR. P. STRICKER.

In the following pages we attempt to describe some points of the natural history and treatment of certain types of arterial obliteration which appear before the age of onset of arteriosclerosis, and are not accounted for by atheroma, syphilis, or diabetes. This study is based on 28 cases, some of which have been followed since 1920.

In spite of the numerous studies lately published on these conditions, they are very insufficiently known. While the pains, the trophic disturbances, and gangrene, which are looked upon as their chief symptoms, are but the final incidents of the disease, we know nothing of its beginning, its mechanism, and its pathogenesis. The mechanism of gangrene and of trophic disturbances looks far more simple than it really is; they are therefore interesting to study. They are sometimes called diseases of evolution, perhaps to prove that to cure them is an impossible task; but this opinion has no serious foundation. At present we must be content to distinguish clearly the precise facts from mere hypotheses and fallacious interpretations.

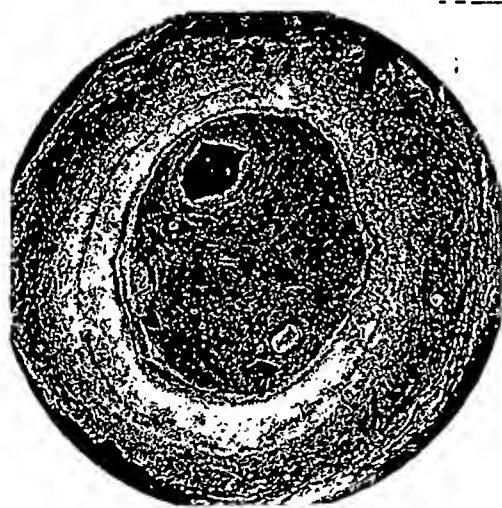


FIG. 395.—Traumatic thrombo-arteritis (due to use of crutches). Gangrene of two fingers. Complete disappearance of pain and vasomotor disturbances two years after resection of axillary artery (10 cm.).

I. THE NOSOGRAPHIC STANDPOINT.

It is necessary to separate four etiological types.

1. *Traumatic Thrombo-arteritis.*

—This is due either to a single traumatism which has caused a contusion of the intima or a complete rupture (closed wound), or to repeated and habitual traumatism —arteritis of those who use crutches (*Fig. 395*). In these cases the clot does not stay in the zone of arteritis. It grows in the vascular lumen, organizes itself, and always produces a coagulation in advance of itself. In this way it reaches

the first large collateral. It stops and may remain in this place or go further on towards the periphery. For instance, we saw the clot depart from the

* Through the kindness and courtesy of Professor Leriche we are able to publish the following account of some of his observations on obliterating arteritis.—Ed. Sec.

ruptured brachial artery, extend beyond the point of bifurcation of the radial and ulnar arteries, and obstruct one of them. At the cardiac end also the thrombosis never remains stationary, but may extend quite a long way. In a case of an occlusion of the subclavian at the level of the formerly fractured collar bone, the clot proceeded further than the interscalenic space. Therefore it is possible that the patient may get worse some months after he is wounded, in spite of his condition appearing to be stationary.

2. *Arteritis from Cold*.—This particularly and almost exclusively attacks the small peripheral arteries, collaterals of the toes, dorsalis pedis artery (*Fig. 396*), and the posterior tibial artery behind the ankle (*Fig. 397*). In these cases the obliteration is frequently not complete.

Many of these arteries with a quite diminished lumen still are permeable to blood. The spastic element here plays an important part in the appearance of the complications. If one wishes to study the circulation of patients formerly frost-bitten, the readings of the classical 'manchette de Pachon' will not be enough. It will be necessary to use a 'manchette' applied about the middle of the foot; otherwise the diagnosis will be missed.

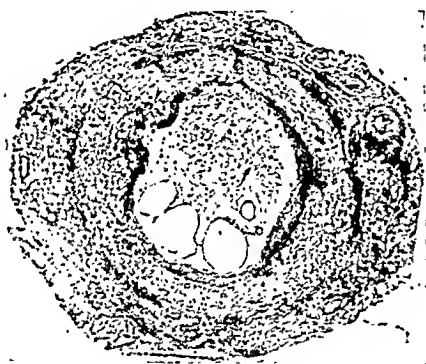


Fig. 396.—Dorsalis pedis artery (arteritis from cold).



FIG. 397.—Posterior tibial artery (arteritis from cold).

It is also always necessary to determine by hot and cold immersion whether there is a spastic element. If after the immersion no modification of the abnormal oscillometric data is seen, one concludes that most of the arteries which go from the leg to the foot are thrombosed. The circulation, which is very precarious, therefore only finds its way through unnamed collaterals. A case has been seen where the peroneal artery was alone responsible for the circulation in the foot.

3. *Localized Monarteritis*.—This condition, which is sometimes seen in adults, though it resembles the thrombosis of embolic origin, has really nothing to do with it. The disease is more subacute than chronic. Its evolution seems to be marked by two periods, one which shows a simple occlusion with sufficient compensatory circulation, and one of gangrene appearing sooner or later. The artery presents a thrombosis of 8 to 10 cm., is distended and blackened by extensive adventitial and peri-arterial oedema. The inflammatory infiltration binds together the collateral veins with the

artery in a single block. It is not an infectious process. Neither the clot nor the walls give positive bacteriologic cultures. There are no infectious signs in the microscopic sections. The patient recovers if the whole thrombosed segment is resected with a ligature round the sound artery. If the ligation crushes the end of the clot, there is a relapse with a progression in the disease. This kind of arteritis is still practically unknown. Two cases have been observed, one of which was followed for three years.

4. *The Thrombo-angiitis Obliterans of Buerger*.—This must not be confused with the other types of chronic arteritis, particularly with arteriosclerosis. It is quite a definite entity, characterized by its early beginning between the ages of 20 and 30, the multiple foci, the bilaterality which is found almost constantly at the first onset, and the venous attacks. On the other hand, Buerger's disease is certainly the same condition as Winiwarter in 1879, Zoege v. Manteuffel in 1891, Heydenreich and Etienne in 1887, and Dutil and Lamy in 1893, had already observed. Therefore it is not

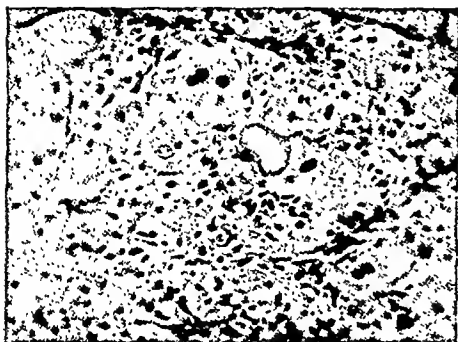


FIG. 398.—Thrombus filling femoral artery, with giant cells which are probably common phagocytic cells attacking particles of coagulated blood.

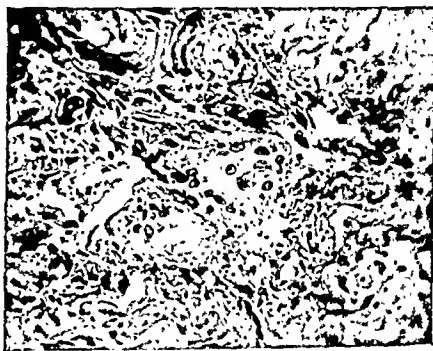


FIG. 399.—Giant cells in the border of the elastic externa of an obliterated axillary artery. Repeated traumatism by crutches. Inclusion of an elastic fibril in the left cell.

a new disease; but Buerger, who does not omit to mention the preceding authors, was the first to individualize the condition, to show its frequency, and to write its whole anatomical description which was previously unknown.

Buerger believes that the disease has an infectious origin, and attaches great importance to the focus of leucocytes and giant cells in the border of the red clot. These injuries are probably not specific. One finds inflammatory foci, more or less important, in the arteries of nearly the whole series of extremities amputated for gangrene, and in numerous arteries surgically removed. Giant cells have been seen in the middle of the clot in an obliterated femoral artery; it appears that they were probably common phagocytic cells attacking remains of coagulated blood (*Fig. 398*). In another case, in an axillary artery thrombosed by repeated traumatism (due to the use of crutches), giant cells could be seen in the border of the elastic externa (*Fig. 399*). Therefore we do not believe that particular significance must be accorded to the details described by Buerger, but the character of the

thrombo-angiitis obliterans remains quite distinctive, and the disease retains its clinical individuality.

II. THE PATHOLOGICAL STANDPOINT.

The different varieties of arterial obliteration look very much alike, and it does not appear that histology can give the key to their problem. While chronic disease of the arteries, depending on disturbances of nutrition, begins at the level of the media by the simple calcification of Moenckeberg or at the level of the elastic interna in the case of atheroma—in other words, in the zones of minimal circulation in the wall—on the other hand, the cases of pre-senile chronic arteritis all seem to begin with an injury of the endothelium. But we believe that nobody has ever seen the first alteration of the walls. Buerger has indeed described early injuries preceding the organization of the clot, but these cases presented otherwise old injuries in other arterial segments.

We only recognize obliterating arteritis when the arteries have been thrombosed for quite a long time. Therefore we see arteries whose thrombi are more or less organized, in some places showing secondary degenerative changes and at others having undergone functional adaptation of the vascular walls.

At the time we observe them, all these cases of obliterating arteritis look alike, because an arterial wall in contact with a clot reacts in a very elemental way. Even after arteriectomy on the macroscopic pieces or on the amputated limbs we are not able to discern the first localization of the disease. At least we have never been able definitely to state "this is the point where the disease began", even after making classified and numbered sections which permitted of the reconstruction of the artery.

Pathology therefore seems to be of little use in the study of the pathogenesis of pre-senile arteritis. Of this we find a new illustration in the study of the arteritis produced by cold. This usually begins in the arterioles by intravascular coagulation, which is the starting-point of arteritis and phlebitis mostly of the thrombosing type. The appearance of an ordinary arteritis is very rapidly produced. The final histological pictures are so similar that numerous authors consider cold and freezing as favouring thrombo-arteritis, without discerning any difference in mechanism at the beginning. *In fact only one anatomical datum is worth considering, and it is more macroscopic than histologic: there is an endo-arteritic arteritis and an arteritis which has become external secondarily.*

In the first category the vascular wall looks normal and the artery is not outwardly changed. In the second the external coat is thickened, adherent, hypervascular, and contains a network of vasa vasorum very much dilated. In addition there may be an intense peri-arteritis binding together attendant veins and sometimes neighbouring nerves. It is not, however, a case of two different types of arteritis—there are simply two phases of the disease. It seems that for a long time the arteritis progresses only in the arterial lumen and in the wall itself, then finally the injuries extend further than the middle coat and reach the adventitia as well as the zone of the nerves and ganglia.

Observation shows that gangrene or trophic disorders seldom occur in the first period; on the other hand, they are usual when the adventitia

is involved. We notice two types of invasion, sometimes by a simple collateral circulation taking the place of the normal nutrition coming from the lumen through the endothelium, which is deficient in this case; at other times by an inflammatory invasion accompanied by oedema and sclerosis. These statements of macroscopic pathology, which are confirmed by more detailed histopathology, are of great importance to the understanding of the clinical disturbances in obliterating arteritis.

III. THE STANDPOINT OF PATHOLOGICAL PHYSIOLOGY.

The clinical and evolutionary history of thrombo-arteritis as a peripheral arterial disease is dominated by one fact, to which no importance has been accorded to this day, except by one of us, who has long insisted on its value.* *Arterial occlusion alone is not sufficient to produce gangrene of extremities, even when extensive, in a main arterial trunk (subclavian, femoral, external iliac), or in an elderly man.*

The only characteristic of the occlusion of a large artery is an insufficient functional circulation—for instance, intermittent claudication in cases of femoral occlusion. This needs to be proved, for at first sight it seems to run contrary to our surgical experience of ligations; but it may be positively demonstrated. First, considering the lower extremities, numerous patients have no pulse, no oscillations on the Pachon, on both sides, but have trophic disturbances and gangrene on one side only.

One of us has previously reported many such examples, and others working on arteritis noticed these cases, which are common in Buerger's disease. Moreover, accounts of post-mortem examinations were published a long time ago in which it was proved that the two ilia communis and ilia externa arteries were obliterated along their whole length without the least trophic disturbance at the level of the lower limbs. Furthermore, experimental demonstration has provided us with definite evidence of the following fact. Threatening disturbances preceding gangrene recede by resection of the whole thrombosed segment; this proves that trophic disturbances and gangrene are not the result of a simple spreading arteritic occlusion.

Since 1916† thirty arterial resections have been performed to combat the disturbances resulting from an occlusion, and we have never seen arteriectomy followed by gangrene. Once only, in a case of unrecognized Buerger's disease, threatening gangrene could not be averted. It is quite obvious that the hydraulics of the main artery is not improved by resection of 7 to 8 cm. of the thrombosed segment. It is more than ever deficient, the removed segment being generally longer than the thrombosed one. Still gangrene does not appear.

Observation 1.—A woman, age 46, from the age of 30 showed diffuse vasomotor disturbances of an upper limb with Raynaud's crises resulting in the appearance of ulcers on some fingers. No oscillations on the Pachon.

* "Mécanisme des Troubles consécutifs aux Oblitérations artérielles spontanées, d'Origine artéritique en dehors de l'Athérome", *Soc. de Chir. de Lyon*, 1924, Oct., in *Lyon Chir.*, 1925, No. 1.

† LERICHE and HEITZ.—"De la Réaction vasodilatatrice consécutive à un Segment artériel oblitéré", *Comptes rend. Soc. de Biol.*, 1917, Feb. 3 and Feb. 17. See also *Bull. et M m. Soc. de Chir.*, 1917, Jan., 310.

On Feb. 12, 1921, resection was made of 7 or 8 cm. of the occluded subclavian artery. All disturbances disappeared, and the ulcers began to heal quickly. In January, 1928, after seven years, recovery was still maintained and there were no more disturbances.

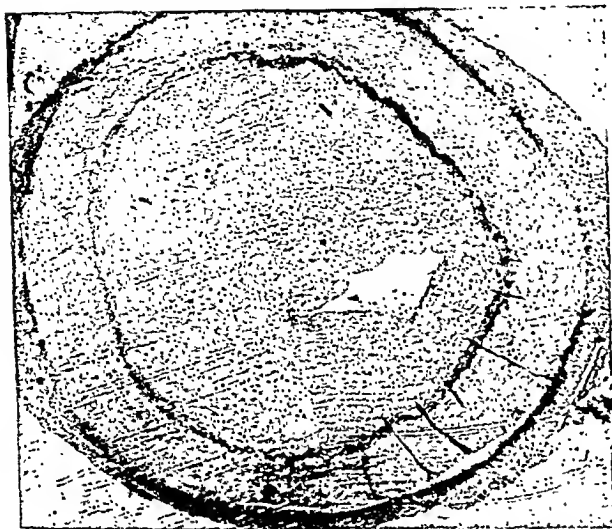


FIG. 400 (Case 3).—Superior part of the resected obliterated femoral artery. Calcification of the media, Mönckeberg type.

2. A man, age 47, who for nine months had noticed cyanosis, coldness of the last three fingers on the left hand, and some pain, suddenly presented gangrene of the last two phalanges of the forefinger and middle finger. On Aug. 9, 1924, resection of nearly 12 cm. of the brachial artery was done. Disturbances receded. The patient recovered without amputation of the fingers. Good

health resulted for eleven months. In July, 1925, one year later, the pains and ulcers reappeared. The point of bifurcation of the radial and ulnar arteries exposed showed a recent clot obliterating the radial and ulnar arteries. The forearm was amputated. The patient was still in good health in January, 1928.

3. A man, age 56, was seen, complaining of painful symptoms and intermittent claudication on the right side, with beginning trophic ulcer on the second toe. There was erythromelia. Very small oscillations were noted on the right side. On May 12, 1927, resection was made of 5 cm. of the thrombosed superficial femoral artery (Fig. 400). Pains and trophic disturbances

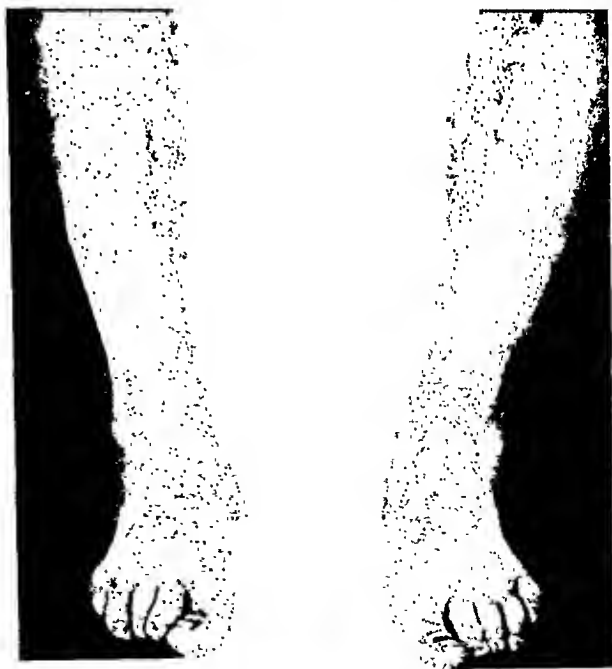


FIG. 401 (Case 3).—Nine months after resection of 5 cm. of the obliterated right femoral artery. Disappearance of all disturbances. The man was free from pain.

disappeared. Later the function of the leg improved and continued so after nine months. In February, 1928, the condition was healthy (*Fig. 401*).

4. A man, age 27, five years after an attack of measles, noticed the appearance of trophic disorders on the third, fourth, and fifth toes of the right foot, with great pain. His fifth toe was amputated for gangrene and he underwent a perifemoral sympathectomy. Recovery, without pain and obvious disorder, lasted four years. Pains have reappeared within the last six months, and when the patient was first seen on June 2, 1928, the foot was very painful, and it was impossible for him to sleep. There were signs of high obliteration. No oscillations in the limb, even just below the edge of Poupart's ligament.

On June 12, 1928, *arteriectomy of the obliterated iliaca externa* was done, and improvement took place the next day. Two months later he was still very pleased, pains had disappeared, he slept well, and was able to cut his nails without pain. The foot was perspiring again and the skin of the toes was more supple; the horny skin on the internal side of the big toe had disappeared.

5. A printer, age 56, in very good health. On Sept. 2, 1928, his left arm suddenly became very painful. At the same time the distal extremity of the limb grew cold. No heart disease, no increase of tension, no infectious maladies, no diabetes. He denied syphilis; the Wassermann reaction was negative. Pains disappeared after hot immersion and the hand again became movable, but the sensation of cold remained. Seventeen hours later the same phenomenon reappeared and immersion was of no use. For a week there was no improvement, then pains in the hand and forearm decreased slightly, and the hand and elbow could be moved.

Obliteration of the lower part of the brachial artery was revealed by palpation and Pachon's apparatus.

Resection of the inferior part of the brachial artery, which was obliterated by red clot to the point of bifurcation of the radial and ulnar arteries, was performed before the members of the Surgical Club visiting Strasburg on Sept. 25. The radial artery was thrombosed, the ulnar artery was free below the bifurcation (*Fig. 402*). Improvement was quite noticeable even on the first day; the hand was warmer and the unpleasant sensation of cold had disappeared. When discharged on Oct. 4, the hand, which was white and cyanotic before the operation, showed an almost normal colouring again. It was warmer than before; the fingers moved very well (*Fig. 403*).



FIG. 402 (Case 4).—Obliteration of the brachial artery and the bifurcation of the radial and ulnar arteries. The resection was performed in the presence of the visitors of the Surgical Club, Sept. 25, 1928.

the forefinger and thumb alone being still a little difficult to move. The apparatus of Pachon showed very small oscillations on the wrist between the divisions 10 and 2, whilst they were formerly altogether absent.

These facts show that in the complications following arterial occlusion trophic disturbances are not caused by suppression of circulation through the diseased artery, but that something more than a simple ischæmia is necessary.* This new element is brought about by vasomotor disturbance. The peripheral arterio-capillary network is nearly always spontaneously distended when an artery is thrombosed over a certain length. The extremities of the patients are usually red in colour. We know that this erythromelia, painful as the limb becomes hotter, stops when it is elevated. The extremities in this position become white as marble, very cold, and look quite bloodless.

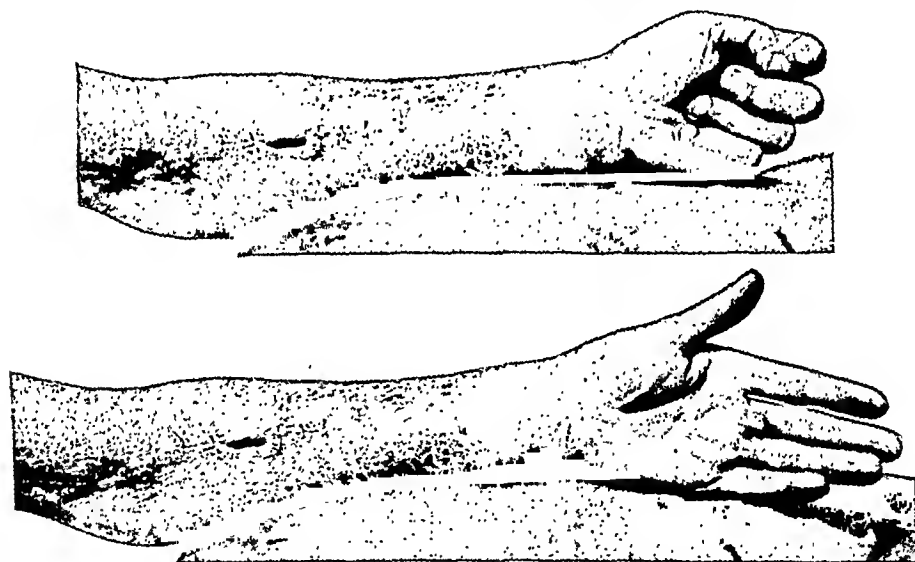


FIG. 403 (Case 4).—Showing the good mobility of the fingers nine days after resection of the brachial artery (see Fig. 402).

The blood-stream is not strong enough to penetrate through the indirect anastomotic channels to the small vessels; it follows the force of gravity.

When the leg is lowered, it at once assumes a bright red hue. Lipiodol having been injected above the occluded segment, X rays reveal a tight network of very thin vessels. Injection into a normal artery never produced such a rich network of arterioles. Consequently vasodilatation exists and provides nutrition for the peripheral tissues when the main normal channel is thrombosed. But this nutrition is precarious, for should the least vasomotor disorder occur owing to traumatism (nail-cutting), strong emotion, or untimely adrenal injection, nutritive disorders start and blood-flow is insufficient to ensure their repair.

* Nevertheless there may be an incomplete necrosis of muscle fibres in the regions where terminal arterioles arise from the main diseased artery, as is the case at the level of the elbow (superior arteries of the flexors). But this has nothing to do with the gangrene of the extremities which we are here considering.

The normal course of the disease may produce the same result. As soon as the intramural process reaches the adventitia, there appear vasomotor reflexes, which work upon the peripheral vessels. These shrink, the circulation, which was formerly sufficient, fails, and gangrene results. Arteriotomy is necessary to prevent this. Moreover, as a general fact, if arterial emboli are quickly followed by gangrene, it is because the clot stops and distends the arterial wall, the externa is therefore forced back, rendered anæmic, and soon shows circulatory disorders and œdema. This is proved by the fact that if, instead of making an embolectomy with suture, the arterial segment is resected, the patient recovers as after the ideal operation, except that the pulse has disappeared. For instance:—

6. A patient, age 47 (latent mitral stenosis), showed suddenly on May 15, 1924, a pain in the left arm with immediate ischæmic disorders. Hand and forearm were as white as ivory, purple stains appeared on the back of the hand, and there was no pulse. The brachial artery was exposed after ten hours. Incision of the artery, extraction of the embolus. The arterial wall was red, velvet, and swollen. Thrombosis was probable: 4 cm. of the vessel were resected; recovery without gangrene.

Therefore we must remember: (1) *That arteries are not merely pipes of distribution, but that all of them are accompanied by a network of nerves.* (2) *That the pathological physiology of thrombo-arteritis cannot be reduced to analysis of the disorders of the circulation at the level of the diseased zone.* (3) *That an occluded artery is no more an artery, but a plexus of sympathetic nerves in an abnormal condition.* (4) *That the nervous element of the adventitia is quite as important as the pipe.*

Because of the frequent alterations of the adventitia and of peri-arteritis, we are bound to think that the addition of adventitial phenomena to the disorders caused by occlusion itself determines the trophic disturbances. It is in fact the endarteritis that has spread through the adventitia which produces gangrene in tissues where the circulation is otherwise just sufficient. The often very paradoxical evolution of many cases of thrombo-arteritis is thus clearly explained.

IV. THE CLINICAL STANDPOINT.

Cases of thrombo-arteritis have generally a latent period in which they are only recognized by those who look for them. They are nearly always undetected before the period of trophic disorders and gangrene. Still they may be easily recognized if oscillometric experiments are made on patients who present painful and feeble legs, intensely cold feet, and especially intermittent claudication. Before the appearance of trophic disorders, cases of arteritis show one or more of the following symptoms: noticeable muscular atrophy of a limb, red foot, blue foot, white foot, foot with œdema, atypical Raynaud's crisis with persistent cyanosis and without restoration of the circulation between the painful paroxysms (the reverse of what appears in real Raynaud's disease). The two most common types begin with intermittent claudication and the appearance of trophic disorders after visiting the corn-cutter. Leg and thigh, forearm and arm, on the four limbs have to

be examined systematically by immersion in hot and cold water. This is quite necessary, otherwise many cases of arteritis may not be recognized. Sometimes instead of oscillations failing, their amplitude will be greatly increased. These cases have nothing to do with arterial thrombosis; often they follow rigidity of the walls in contact with a periphlebitic sclerosis.

V. THE THERAPEUTIC STANDPOINT.

It may be stated that if the artery is anatomically occluded and not spasmodically contracted, no indirect operation on the nerves will be of use at any period of the obliterating arteritis.

Sympathetic operations, peri-arterial sympathectomy, and ramisections should be undertaken only in cases of incomplete obliteration of dilatable vessels, and if there is no compensatory vasodilatation or lymphangitis in the periphery. This often occurs with arteriosclerotic individuals and sometimes with frostbitten cases whose arteries are always thickened and not always obliterated; in general whenever the spasmodic element plays the chief part in the symptomatology. This may be also the case at the beginning of Buerger's disease, when obliteration is still slight. The surprising successes brought about by Hartmann, Georges Muller, Archibald, and Bernheim by peri-arterial sympathectomy, which were quoted at the last Surgical Congress, are thus explained. It is generally too late, however; obliteration has spread and the time for sympathetic operations is past. Since 1923 they have only been tried in cases which looked propitious or when compelled by threatened amputation; but with no success. Other nerve operations like cordotomy are still more inefficient. Cordotomies were sometimes done following false diagnosis in cases of pains not referred to their real cause. Results were not satisfactory. Only two operations appear of any use in obliterating arteritis: (1) Arterectomy; and (2) Epinephrectomy.*

1. **Arterectomy.**—Eleven years of practice and the investigation of thirty cases show that arterectomy may be considered when a large artery alone is obliterated or when several small peripheral arteries are concerned. In fact it is but a *sympathectomy made exactly at the diseased point* so as to abolish the zone of abnormal departing reflexes. It is followed by the same vasodilator reaction as peri-arterial sympathectomy, as may be found recorded in a paper read before the Société de Biologie.*

In mono-arteritis recovery is generally lasting. In traumatic arteritis, whatever its mechanism, the patient is greatly improved. The extremity gets warmer, pains and trophic disorders disappear, and oscillations may sometimes be seen on the Paehon apparatus. Abolished voluntary movements are again possible; the result is lasting. Twenty-two cases have been observed, but an application to cases of declared gangrene would be obviously too optimistic. In spontaneous arteritis localized to only one artery without tangible cause, arterial resection is the only immediately efficient treatment and the only method which can perhaps give a lasting result. For instance:—

* LERICHE and HEITZ.—“Influence de la S.P. et de la Résection d'un Segment artériel oblitéré sur la Contraction volontaire des Muscles”, *Comptes Rend. Soc. de Biol.*, 1917, Feb. 17.

Observation 7.—A man, age 36, after having stayed in water for several hours, suddenly felt pains in the right arm, in the neck near the shoulders, and general discomfort. The next day the right arm was painful, heavy, and could hardly be moved. Pains spread from arm to fingers, and were then located in the forefinger. There were



FIG. 404 (Case 7).—Resection of the obliterated brachial artery (9½ cm.).

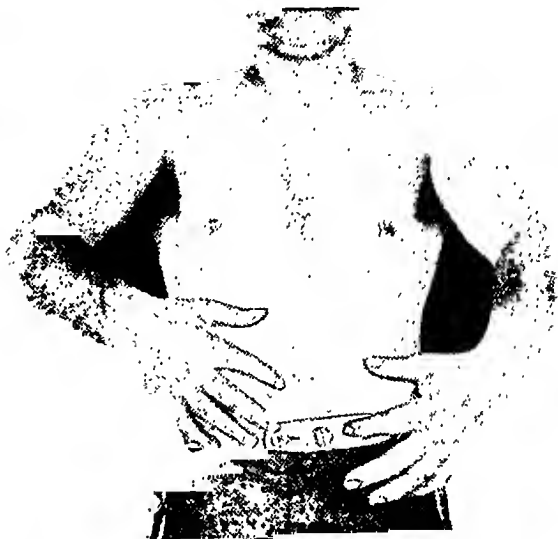


FIG. 405 (Case 7).—Five months after resection of brachial artery (see Fig. 404).

also suffocating crises which warmth increased. Three months later the patient was sent to hospital with the diagnosis of Raynaud's disease. Accordingly a low ramisection was made on Nov. 7, 1926. Excellent immediate result, followed by recurrence in January, 1927. On account of this, humeral sympathectomy was performed on an artery found very contracted. The condition improved, but in August, 1927, recurrence took place. The patient asked for an amputation. He suffered too much and was too impatient. At the end of August the brachial artery was resected from the lower third to the anterior fold of the axilla, near the first collateral (Fig. 404). It was completely obliterated by an old thrombus in the middle and by a recent one at both extremities. Large adventitial injuries. Immediate improvement.



FIG. 406 (Case 7).—Five months after resection of brachial artery.

In October the armpit was still painful, where the thrombus appeared to progress; but it soon stopped, and the last news was that the patient had quite recovered. He came to hospital in December and in January riding a bicycle; his right hand was as good as the left and nearly the same as before his illness. He has fattened and works regularly. Still, he has no brachial artery from armpit to elbow, nor any radial pulse. (*Figs. 405, 406.*)

Arteriectomy is the only operation which relieves cases of old frostbite, when it is really a question of arterial thrombosis and no mere diminution of calibre with superimposed spasms—which may be found out by oscillometry and hot immersion.* It is rather strange to see ulcers healing and the foot getting warmer after resection of the posterior tibial artery and dorsalis pedis arteries. Can this result be lasting? It has been observed for several months; but a recurrence is to be expected, for generally patients are seen after years of pains and various circulatory disorders. Since, with the exception of arteriectomy, amputation of the foot is the only possibility, arteriectomy may in any case be attempted.

Arterial resection was tried in Buerger's disease more than once. It had only passing sedative effects. It is of no real value except to establish the diagnosis. On the other hand, in this disease great improvements are obtained by unilateral epinephrectomy.

2. **Epinephrectomy.**—It seems needless to say that complete recovery may not be expected when the two femoral arteries are transformed into fibrous cords. It is plain that signs of ischæmia will persist whatever may be done, for the damage cannot be repaired. Intermittent claudication is evidently incurable; but one can attempt to obtain the abolition of pain with improvement of the peripheral circulation, and especially to arrest the evolution of the disease. This result was obtained in many cases.

Up to now thirteen epinephrectomies have been performed in different vascular diseases, nine of which were for generalized obliterating arteritis; six of these cases looked like Buerger's disease. The first seven observations have been published.† These are the actual results:—

One patient was very much improved by the operation for seven months, but successive amputations of the two legs, one in a year, the other two years after epinephrectomy, were unavoidable.

One patient, operated on in February, 1926, has shown since epinephrectomy some trophic disorders of the foot. Intermittent claudication still remains, and pains have disappeared; there is no gangrene and amputation was not necessary. His general status is quite good, he goes every day to his office and continues with his work. Operation was done two years ago.

A third patient, a doctor, was operated on in December, 1925; he arrived on a stretcher, and went away walking, using both legs, though

* In this case peri-arterial sympathectomy may give lasting results. Instances are to be found in the reports of the French Congress of Surgery, 1927.

† LERICHE.—“De la Surrénalectomie dans les Gangrènes artérielles des Sujets jeunes”, *Soc. de Chir. de Lyon*, 1925, Dec. 10, and *Bull. et M. m. Soc. de Chir.*, 1926, May 12, No. 17, 521.

STULZ and STRICKER.—“Huit Observations de Surrénalectomie dans l'Enartérite oblitérante juvenile et dans la Maladie de Buerger”, *Rev. de Chir.*, 1927, No. 3, 196.

an amputation had at first seemed to be necessary. After eighteen months he wrote that his recovery was progressing regularly and that he would soon begin again with his work.

In a fourth case the operation failed, and amputation of the thigh was necessary; since then nothing has been heard of the patient. A fifth, a dentist, was operated on in March, 1927; he could not work over an hour without being obliged to lie down; since epinephrectomy he has been quite well. His leg was not amputated, he shows no trophic disorders, and now works standing for periods up to six hours. In a last case, operation is still recent and the result doubtful.

To sum up: 2 failures, 1 improvement followed by a recurrence, and 3 very good results lasting from one to two years.

If patients and their condition before operation are considered, we must acknowledge that epinephrectomy is useful and its study is to be continued. Coincidences and spontaneous recovery may be easily invoked in successful cases. But as one has seen epinephrectomy followed by sudden disappearance of pains and quick recovery from trophic and gangrenous disorders in patients who could not be relieved and had been previously more than once uselessly operated upon, one must continue to believe in the therapeutic value of this operation.

Last year Costantini proposed ablation of the medullary part alone, which is easily done with dogs, in place of epinephrectomy. This very logical operation has been tried with men twice without any success, for the capsule is extremely friable. Anyhow it seems that these attempts may be continued, and that, thanks to epinephrectomy and arteriectomy, endarteritis will appear in the future not so hopeless as it is to-day. Amputation is no longer the only treatment,

REVIEWS AND NOTICES OF BOOKS.

Addresses on Surgical Subjects. By SIR BERKELEY MOYNIHAN, Bart., K.C.M.G., President of the Royal College of Surgeons of England. Medium 8vo. Pp. 348, with 12 illustrations. 1928. Philadelphia and London: W. B. Saunders Co. 28s. net.

WE are glad to have the opportunity of noticing this collection of orations and addresses, although we think that the majority of them have already been read by most English surgeons. We realize in reading these essays that surgery is to their author not merely a science or an art but a religion. It is indeed a happy fact when the highest exponent of our art has the burning eloquence to extol it. Of the fourteen chapters which the book contains, more than half are devoted to a critical review of the leaders and teachers who have gone before. Hunter and Lister, the founders of our present knowledge and ideals, take the pride of place; the old teachers from Hippocrates to Galen and from Galen to Hunter have their work reviewed; and the men of our own generation, some of whom have only lately passed away, such as John B. Murphy, Mackenzie Davidson, and the surgeons of the Leeds School, have a generous appreciation given of their work. It is remarkable the wealth of eulogy that the orator has to bestow upon nearly all those whom he mentions, and what a wonderful word portrait he is capable of drawing of the men he has known personally.

A number of the essays are devoted to those special subjects of abdominal surgery with which the name of Moynihan has become so closely associated—those of the stomach, duodenum, pancreas, and gall-bladder. Probably the essay which might most lay itself open to sceptical criticism is that dealing with the relation of aberrant mental states to organic disease; but, after all, it is pleasant to be stimulated to occasional disagreement as well as to be carried away by compelling eloquence.

Recent Advances in Surgery. By W. HENEAGE OGILVIE, M.A., M.D., M.Ch. (Oxon.), F.R.C.S. (Eng.), Assistant Surgeon, Guy's Hospital, and Lecturer in Clinical Surgery to Guy's Hospital Medical School, etc. Crown 8vo. Pp. 461 + viii, with 108 illustrations. 1928. London: J. & A. Churchill. 15s. net.

As the author points out in his preface, it is not an easy matter to decide what should be included under 'recent advances' in surgery—by the time a suggested method has been thoroughly tested and genuinely accepted as an 'advance' it has ceased to be recent, and many recent 'advances' have been in a retrograde direction. This is especially so in connection with the surgery of gastric and duodenal ulceration, and the author's aphorism, "Every operation for ulcer appears to be a success until it is found out", is delightfully naïve.

Few will fail to agree that Mr. Ogilvie and his co-writers have made an extremely wise selection of 'advances' from the enormous mass of material available, and have shown a most judicious and common-sense attitude in discussing the relative values of the various procedures described. With the exception of the chapters on cancer by J. F. Carter Braine, on plastic surgery by T. P. Kilner, on chest surgery by Grant Massie, and on venereal disease by V. E. Lloyd—all of them most excellent—Mr. Ogilvie is responsible for the whole of the book, and is to be congratulated on a truly great achievement.

Most practising surgeons will agree with the opinion that although fractional test-meals are useful in deciding the nature of the treatment in cases of gastric and

duodenal ulcer and the progress of the case under such treatment, yet for diagnostic purposes the evidence they yield is much inferior to that given by radiography, and is hardly commensurate with the expenditure of time and labour which they necessarily involve.

The chapter on plastic surgery represents work developed almost entirely during and since the war, and a careful perusal of the marvellously ingenious operative procedures therein detailed conveys the impression that plastic surgery is only in its infancy—an impression strangely at variance with the statement in the introduction, with which Sir Berkeley Moynihan apparently agrees, that "Surgery as a mechanical craft is almost complete."

There is a sufficiency of good illustrations, and at the end of each chapter are given numerous references to recent surgical literature—a feature which should prove most useful and time-saving to a busy surgeon. Altogether, the book attains a very high standard and admirably supplies a very real need.

An der Schwelle der Chirurgie. By EMILE FORGUE, Professor of Clinical Surgery in the University of Montpellier. Translated by Professor Dr. GEORG SCHMIDT, Munich. Medium 8vo. Pp. 136. 1928. Leipzig: F. C. W. Vogel. Paper covers, M. 10; bound, M. 11.50.

A BOOK written by a distinguished French surgeon which has merited translation by a German surgeon must necessarily attract our attention. In reading it our expectations are not disappointed, because from the first to the last page it is filled with sound wisdom, the fruit of ripe experience, expressed in the happiest of language. It is concerned with various aspects of surgery, chiefly ethical, and is dedicated to the younger generation of surgeons. It begins with advice to students whose ambitions are to become surgeons, and this section is concerned chiefly with the need for technical dexterity, guided by careful clinical diagnosis. Other chapters follow on "Empiricism and Surgery", "The Relation of Surgery to Medicine", "Honour before Life", "The Ethical Laws of Surgery", "The Responsibility of the Surgeon and Euthanasia". Throughout all these essays, which have been delivered on various special occasions, there are exhibited the results of great surgical experience, philosophical learning, and a very wide knowledge of foreign literature. The motto of the German Surgical Society, "Truth in Science and Conscientiousness in Art", is repeated more than once, and made the text of much sound advice.

The last chapter, on euthanasia—or, as it is explained to mean, 'glorious death'—is one of the most intriguing of all the essays, containing as it does not only the scientific discussion of dissolution, but also very copious references to the death-bed experience of well-known historical characters, such as the alleged utterance of William Hunter: "If I had but the strength to hold a pen I would describe how pleasant and easy it is to die." This subject naturally ends with an eloquent tribute to those of the younger generation of surgeons and students who found a glorious death in fighting for their country. In conclusion, we can only express a sincere wish that these addresses may be translated into English.

The Robert Jones Birthday Volume: A Collection of Surgical Essays. With a Preface by Sir BERKELEY MOYNIHAN, Bart., K.C.M.G. Crown 4to. Pp. 434 + xii, illustrated. 1928. London: Humphrey Milford. 42s. net.

THIS volume, to which the leading orthopædic surgeons in Britain, and one or two besides, have contributed papers, forms a very handsome and valuable tribute to the esteem in which Sir Robert Jones is held in the surgical world. In the preface Sir Berkeley Moynihan voices in characteristic language the debt which surgery owes to Jones. The list of contributors is a striking one and contains the names of many distinguished surgeons, one and all of whom acknowledge in Jones their teacher and their friend.

It may be said at once that this book represents in a signal manner our present-day knowledge of orthopædic surgery, and that it is a most valuable collection of papers by experts, each writing on a subject which he has made his own. So high is the standard of excellence that it would be invidious to select single papers for special comment, except to indicate the broad field which is covered.

The opening paper on "Orthopædics before Stromeyer" gives a most interesting and well illustrated account of the beginnings of modern orthopædic surgery, with particular notes on the lameness of Scott and Byron. Osgood, of Boston, contributes a paper on the "Association of Intestinal Stasis with Spinal and Sacro-iliac 'Arthritis'". Since Lane laid perhaps undue stress on the evil effects of intestinal stasis, there has been a reaction and a tendency to underrate the importance of this as a factor in the etiology of chronic arthritis. Osgood's paper brings fresh evidence in favour of Lane's views. The paper on "Dissociation of Bone Growth" by Murk Jansen is a long and thoughtful one which will be read with great interest, and that on "Congenital Dislocation of the Hip" by Hey Groves is a terse but most complete study of the pathology of this condition and its treatment on rational lines. The value of both these papers is enhanced by admirable illustrations. Allison, of Boston, writes on "The Open Operation for Congenital Dislocation of the Hip"—an excellent presentation of the subject.

Of the remaining papers, no one of which should be left unread, special mention must be made of Elmslie's striking article on "Fibrocytic Diseases of the Bones", Platt's paper on "Peripheral Nerve Complications of Fractures and Dislocations of the Elbow", Fairbank's on "Infantile or Cervical Coxa Vara", Girdlestone's on "Operation for Tuberculosis of the Hip", and Laming Evans's on "Astragalectomy".

Sir Robert Jones may well feel proud of his Birthday Volume, for it contains a series of papers which not only represent what is best in modern orthopædic surgery, but also bear the imprint of his teaching and reflect the esteem in which he is held among surgeons. The editors and publishers are to be congratulated on this handsome volume, which should be read by surgeons the world over with the greatest interest.

Diagnostische und therapeutische Irrtümer und deren Verhütung. Chirurgie.
 Edited by Professor Dr. J. SCHWALBE, Berlin. Royal 8vo. Pp. 347, with 140 illustrations. 1928. Leipzig: Georg Thieme. Paper covers, M. 24; bound, M. 26.

THE present volume is only a part of a very large series of works dealing with errors in diagnosis and treatment. The series comprises no fewer than sixteen works on internal medicine and twelve on surgery. The present volume contains articles by Professor Heller, of Leipzig, on the diaphragm, internal hernias, and the intestines; by Professor Hohlbaum, also of Leipzig, on the stomach and duodenum; and by Professor Klein Schmidt, of Wiesbaden, on intestinal obstruction.

It is a very carefully written and complete surgical treatise dealing with both simple and complicated problems of diagnosis and treatment. For example, Professor Heller gives a very full account, illustrated by many radiograms, of the diagnosis between hernia through the diaphragm and paralytic relaxation of the latter. Among the diagnostic methods in common use is the inflation of the peritoneum with air, the parts being subsequently submitted to X-ray examination. In discussing the treatment of chronic constipation the author lays great stress upon the uselessness of operations done for functional conditions of a spastic or atonic character. In the article dealing with hæmorrhoids it is pointed out that the most serious error in treatment is that of producing a stricture of the anus, but we do not find adequate recognition of the value of injection treatment as a simple procedure that is free from risk. There is a good account of anal fistulæ, and careful directions are given for avoiding the injury to the sphincters which produces incontinence. The subject of the diagnosis of ulcer of the stomach and duodenum is dealt with at some length, but we think that its value would have been increased if it had been illustrated by X-ray pictures. The rare conditions of volvulus of the stomach and also of intussusception of the stomach into the duodenum are well described. There can be no doubt that the work is one of great value, being filled

with careful descriptions and critical discussion. If it has a fault, it lies in the direction of being too detailed and of dealing with conditions that are very rarely met with.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, B.A., M.D., M.S., (Lond.), F.R.C.S., (Eng.), Senior Surgeon to Out-patients, St. Mary's Hospital, Paddington, etc. Fifth edition. Demy 8vo. Pp. 244 + xiv, illustrated. 1928. London: Humphrey Milford. 10s. 6d. net.

THIS useful little book has now reached its fifth edition—a fact which alone illustrates the help it has given to members of the medical profession. There are few alterations in the book except that the author has amplified his account of the symptoms of peritonitis and has given his views as to their causation and variation. There is in addition a small section on certain retroperitoneal conditions which may simulate acute abdominal disease, and a few paragraphs dealing with obstruction in the small intestine. The symptoms of acute peritonitis are divided into two groups, reflex and toxic; the reflex symptoms being earlier in onset when the demonstrative part of the peritoneum is affected and delayed in infections of the non-demonstrative areas. Failure to recognize this may lead to delay in the diagnosis of a pelvic or central peritonitis, in both of which non-demonstrative areas of peritoneum are affected. Muscular rigidity, a very common sign, is only found when the demonstrative areas of peritoneum are involved. The important point that it is not present in pelvic peritonitis is emphasized. It may also be absent in people with fat abdominal walls and feeble muscles, or in the aged and weak. A description of the signs and symptoms of pneumococcal peritonitis is given, and it is noted that this may not give rise to muscular rigidity even in its early stages. With this we entirely agree.

In the chapter on acute intestinal obstruction stress is laid on the importance of the involvement of the mesentery. Two groups of obstruction are described, one in which the mesentery is involved—in this the onset is more sudden and the symptoms are more severe and may lead to early peritonitis—and the other in which the mesentery is not involved, the onset being more insidious and the abdominal signs less marked. In the chapter on the signs and symptoms of perforation of gastric and duodenal ulcers we note that the author says the patient is writhing in agony during the stage of primary shock. This is inaccurate and misleading. It is our experience, based upon close observation of many cases at the moment of perforation, that in this stage the patient lies absolutely still and rigid, fearing to move in the slightest degree lest the pain be increased, and we think this represents the true picture from the very onset of the perforation.

One of the most useful chapters in the book is the last, which describes very shortly the signs and symptoms of diseases which may simulate acute abdominal conditions. One of the commonest mistakes in diagnosis of the acute abdomen is the confusion of its signs and symptoms with those of thoracic lesions. A very useful table is given setting out and comparing the signs and symptoms in each case.

The Treatment of the Acute Abdomen, Operative and Post-operative. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S. (Eng.), Senior Surgeon to Out-patients, St. Mary's Hospital, Paddington, etc. Second edition. Demy 8vo. Pp. 244 + xviii, with 146 illustrations. 1928. Humphrey Milford. 10s. 6d. net.

IN the second edition of this book the author has made a few minor alterations, and has also inserted a short account of the work of R. B. Williams and of Brockman on the treatment of the toxæmia of ileus and intestinal obstruction. Post-operative ileus is one of the most dreaded complications of acute appendicitis, partly on account of its insidious mode of onset and partly because of its high mortality. The usual signs and symptoms of this condition are enumerated, and the treatment, non-operative and operative, is described. In the former the work of R. B. Williams on the treatment by the injection of anti-gas-gangrene serum, and that of Brockman,

who injected diluted human or ox bile into the rectum to make up for the loss of entrance of normal bile into the intestine, are shortly described. We have tried both methods of treatment, but not in a sufficiently large number of cases to enable us to draw any definite conclusions as to their efficacy. These methods are, however, such as should undoubtedly be given further trial. The various methods of operative treatment are described, and the author considers that jejunostomy with or without lateral anastomosis produces the best results.

The book is well illustrated, clear, and definite in its description of the different methods of treatment, and is one which will undoubtedly prove of value to those for whom, in the preface of the first edition, the author says the book is written.

Vademekum der speziellen Chirurgie und Orthopädie für Ärzte. By San.-Rat. Dr. HERMANN ZIEGNER. Ninth edition. Medium 8vo. Pp. 326 + vi. 1928. Leipzig: F. C. W. Vogel. Paper covers M. 13; bound, M. 15.

IT is rather difficult to classify this book. It represents a very successful attempt to collect together in the form of short notes the most important practical points in every surgical problem that may present itself in general practice. It is therefore a book for reference rather than for reading, and it must presume access to or knowledge of larger text-books, because all kinds of apparatus and operations are referred to which are not illustrated or explained. A good many of the sections seem to suggest preparation for an examination rather than instruction for a practitioner. For example, the treatment of an ununited fracture is given under two headings and a choice of six methods, but with practically no indication as to the selection of a particular procedure for a particular case. There can be no question, however, about the book's being closely filled with accurate information, so arranged and classified as to make it easy for reference. The fact that it has already reached a ninth edition in a country where new text-books of surgery appear every year is the best evidence that it has proved acceptable to those for whom it was written.

A Text-book of Fractures and Dislocations, covering their Pathology, Diagnosis, and Treatment. By KELLOGG SPEED, S.B., M.D., F.A.C.S., Associate Professor of Clinical Surgery, Rush Medical College, University of Chicago; Associate Attending Surgeon, Presbyterian Hospital, etc. Second edition, enlarged and thoroughly revised. Medium 8vo. Pp. 952 + xiv, illustrated with 987 engravings. 1928. London: Henry Kimpton. 50s. net.

THE second edition of this monograph has grown to 952 pages, and a good deal of important information has perforce to appear in small type. The work is undoubtedly based on the considerable practical experience of the author in fracture surgery, which is well exemplified in the many excellent line drawings of radiograms of actual fractures, and the sound advice offered in the treatment of various types of bone and joint injury. But when judged either as an exposition of surgical teaching or as a comprehensive text-book of reference, it suffers in comparison with the standard works. In almost every chapter the 'setting' is patchy; there is no clear enunciation of principles followed by a logical development in sequence of methods of treatment and results. There is too often a strange lack of perspective; thus twenty-seven pages are devoted to that uncommon injury 'traumatic dislocation of the hip-joint', which is presented with all the anatomical minutiae of the old-time surgeon. On the other hand, important and difficult complications are somewhat cursorily dealt with—for example, peripheral nerve injuries accompanying dislocations of the shoulder-joint and elbow fractures. The account of the pathogenesis of Volkmann's contracture is also vague; this subject has been much illuminated by modern experimental work which has a definite clinical application. In the treatment of this deformity the illogical operation of lengthening the individual tendons at the wrist is advocated.

The chapter on carpal injuries—a subject on which the author is a recognized authority—does not do justice to his exceptional knowledge and experience. The over-elaborate classification adopted here could not possibly remain in the memory

of the most brilliant student for more than twenty-four hours, and is in striking contrast with the simplicity of Destot's exposition of the same group of injuries.

On page 354 the dangerous *hyperflexion* position of the elbow after the reduction of a supracondylar fracture is illustrated, with the forearm bandaged closely to the upper arm. In the text it is stated that each day after the reduction the position should be released and that the patient should actively extend the elbow. It may not be the author's intention, but such advice encourages the use of early movements in this type of injury—a proceeding which is always disastrous. In the treatment of ankle fractures the Delbet ambulatory plaster is not mentioned. In spite of such blemishes, however, the book contains much valuable material for the consideration of the experienced fracture surgeon.

A Text-book of Orthopedic Surgery for Nurses. By PHILIP LEWIN, M.D., F.A.C.S., Associate Professor of Orthopedic Surgery, Northwestern University Medical School, Chicago, etc. Demy 8vo. Pp. 353, with 340 illustrations on 161 figures. 1928. London and Philadelphia: W. B. Saunders Co. 15s. net.

This book is intended to give a general view of orthopædic surgery to nurses who have to carry out its principles. The author says: "My purpose is not to bring the nurse to the operating room and close the door in her face, but to take her inside; take her to the ward where orthopædic cases are treated, to the plaster room, to the splint room, and to the brace shop, and make her work in these departments interesting to her and beneficial to the patient."

The most important chapters for the nurse are those on plaster-of-Paris technique, the care of a child with a tuberculous joint, infantile paralysis (especially the prevention of deformity and muscle re-education), and operating-room technique. The book is profusely illustrated, and our only criticism is that perhaps an attempt has been made to compress too much in a small space. There can be no doubt that, as so many of the chronic crippling diseases of children—such as poliomyelitis and tuberculosis—must be treated for long periods of time in sanatoria, where they are in daily contact with the nursing staff, it is a matter of great importance that the latter should understand the general principles of disease and the technical methods used in its treatment. We think that this book admirably fulfils this object.

Les Arthrites gonococciques. By HENRI MONDER, Professeur agrégé de la Faculté de Médecine de Paris; Chirurgien des Hôpitaux. Medium 8vo. Pp. 527, with 121 illustrations. 1928. Paris: Masson et Cie. Fr. 70.

IN this work the author has collected not only his personal observations but also a vast amount of material from the literature of gonococcal joints. Without hesitation it may be stated that the volume contains more information on this subject than does any other work in existence. But whilst one cannot fail to admire the industry and enthusiasm of the man who has collected together so much material, the perusal of a book such as this leaves the reader with a feeling of depression. After all these researches and all this reading in libraries, how little real knowledge do we possess! The chapter on treatment, which is the longest in the book, is the gloomiest of all. The author passes in review every therapeutic measure that has ever been applied in the hope of curing a gonococcal arthritis. In each case he quotes the words of some exponent of the measure in question, and then proceeds to demonstrate that in more cases than not this form of treatment is likely to fail. He brings out the fact that there exist many forms of gonococcal arthritis which are resistant to all therapeutic weapons. Whatever be used—whether it be vaccine therapy, diathermy, radiant heat, serum therapy, congestion treatment, or massage—the condition of the joints becomes progressively worse and ends in destruction of the cartilages and ankylosis.

In contradistinction to such an authority as Robin, who considered that gonococcal arthritis was eminently a medical complaint to be treated by medical methods alone, the author places considerable reliance on surgery as a means of

preventing these disastrous results. He recalls the fact that to Lister must be accorded the merit of having first introduced drainage of joints. In those cases which do not respond to medical treatment, and which appear to be progressing towards destruction of the articular cartilages and ankylosis, he urges that arthro-tomy is the right method of treatment. The chief difficulty likely to be encountered in carrying this out is that of maintaining free drainage, and he describes various techniques that have been employed to overcome this difficulty.

It is somewhat surprising that in a work of this completeness scant attention has been paid to the subject of arthrectomy. Actually there are cases of ankylosis in a bad position for which arthrectomy is the one and only method of treatment; particularly is this so in the case of the knee-joint, a joint which more than any other is likely to be the seat of an intractable gonococcal infection. If the author thinks, as he appears to do, that gonococcal joints are at least as amenable to surgical treatment as are tuberculous ones, surely he must consider that arthrectomy has a place in such surgery.

The volume is well illustrated with photographs and skiagrams. Many of these strike the keynote of the book by illustrating failures in treatment. Most valuable of all, because of its completeness, is the bibliography that concludes the work. The book is not likely to be used otherwise than as a work of reference—it is too long and too negative in its attitude to suit the tastes of the average practitioner or even surgeon; but the mass of material contained within its covers, and the comprehensive bibliography, render it a valuable work of reference.

Konstitutionspathologie in der Orthopädie. By Dr. BERTA ASCHENER, Vienna, and Dr. GUIDO ENGELMANN, Vienna. Medium 8vo. Pp. 312 + vii, with 80 illustrations. 1928. Vienna and Berlin: Julius Springer. M. 28.

THIS book is of a special and rather unusual character. It deals with the underlying causes of deformities. An introduction gives a review of the modern doctrines of heredity, which, as it is in a somewhat condensed form, is a little difficult to understand. This is followed by a consideration of intra-uterine causes of defects in the child, of which pressure of the uterine wall and anomalies of the amnion play the chief part. The authors are not prepared to receive Murk Jansen's theories as reasonable. The body of the work consists in a discussion of various deformities in relation to the possible causative factors. In this consideration a very large space is devoted to the hereditary and familial aspects of these affections.

The different forms of perverted growth, dwarfing, and gigantism are considered in detail, but especially in relation to their causation. The systematic diseases of the skeleton, such as Paget's disease and osteitis fibrosa cystica, receive very short consideration, but these two conditions are regarded as essentially the same. We think that the authors might well increase the attractiveness of their book by giving a larger proportion of illustrations as a relief to the exceedingly difficult problems of heredity and causation which are their chief themes. The pictures of a family suffering from dysostosis cleidoocranialis hereditaria, for example, give the reader a happy breathing-space in the midst of a most difficult volume. The work concludes with an extensive list of papers on the literature of the subject. As a book of reference on an obscure subject it is both useful and valuable.

Plaies et Maladies infectieuses des Mains. By MARC ISELIN, Ancien Interne des Hôpitaux; with a Preface by CH. LENORMANT, Paris. Medium 8vo. Pp. 218, with 66 illustrations. 1928. Paris: Masson et Cie. Fr. 30.

THIS book is a clearly written and well illustrated treatise on wounds of the hand, and, whilst there is little original matter, the whole subject is presented in a most readable form. The part dealing with infections is based largely on the well-known work of Kanavel, and follows faithfully the principles which he has laid down. Some of the methods described for uniting tendons and for making an artificial thumb from the forefinger are well illustrated; these are not generally known in this country.

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For those desiring a short guide to the treatment of hand injuries the book can be recommended. It does not, however, in any sense replace the larger works on this subject which are now well known.

Konservative Frakturenbehandlung. By Dr. LEOPOLD SCHÖNBAUER, Assistant in Surgery to the University Clinic, Vienna. Royal 8vo. Pp. 266 + viii, with 119 illustrations. 1928. Vienna: Julius Springer. Paper covers, M. 16.50; bound 18.60.

ANY work proceeding from the clinic of von Eiselsberg in Vienna at once commands our attention, owing to the very large amount of clinical material and the high traditions which obtain in that school for teaching. The present volume is rather difficult to place among the many recent books which have appeared on the subject of fractures. It is too small and brief to be in any way comprehensive, and it does not contain anything which is new or original. Even the short space devoted to this large subject is partly occupied by a consideration of fractures of the skull and the nerve complications thereof, which is a subject so entirely different from that of fracture of the long bones that it would be better dealt with in another volume. However, as a short and simple description of the conservative methods of treatment used in Vienna it is useful and instructive. The author takes a very reasonable view of the indications for operative treatment. He states that in simple fracture open operation restores the patient to functional activity more quickly than conservative in cases of the patella and the olecranon, but not in the case of the long bones. We are interested to find that Hackenbruck's apparatus is still largely in use; it consists of a distraction apparatus fastened to the limb by plaster-of-Paris. We think that the book would be improved if the references to operative treatment were entirely omitted, because such references are either too short to be any use or else rather misleading—for example, open operation for a fractured clavicle is described and figured, although probably there is no long bone in which such an operation is less frequently required, and the method which is actually depicted is a most inefficient fixation with a single loop of wire.

The letterpress is clear and concise and the illustrations are well chosen and well reproduced. There is a commendable recognition of the work of surgical schools outside Germany.

Chronic (Non-tuberculous) Arthritis: Pathology and Principles of Modern Treatment. By A. G. TIMBRELL FISHER, M.C., F.R.C.S., Late Hunterian Professor, R.C.S., England; Surgeon with charge of Out-patients, the Seaman's Hospital, Greenwich, etc. Demy 8vo. Pp. 232 + ix, with 186 illustrations and 93 plates. 1928. London: H. K. Lewis & Co. Ltd. 25s. net.

THE author of this small work, who has become a well-known authority upon the surgery of the joints, justifies the appearance of this new volume by presenting the subjects of osteo-arthritis and rheumatoid arthritis in relation to the physiology of the joints, and to experimental investigation. The great and most commendable feature of the book is the manner in which it is illustrated. Most of the drawings are from specimens in the Hunterian Collection or in the museums of the London hospitals. As an exposition of the pathological changes which the joints undergo in chronic arthritis, both macroscopic and microscopic, the book holds a very high place; in fact we know of no treatise of the same size in which there is such a good collection of illustrations so beautifully reproduced. A large number of these have already appeared in the *BRITISH JOURNAL OF SURGERY*, a fact which is acknowledged by the author in the Preface. The disappointing feature of the work is the comparatively meagre sections which are devoted to treatment.

Requisites and Methods of Surgery. By CHARLES W. CATHCART, C.B.E., M.A., M.B., C.M. (Ed.), F.R.C.S. (Eng. and Ed.), Consulting Surgeon, Royal Infirmary, Edinburgh; and J. N. JACKSON HARTLEY, O.B.E., M.B. (Ed.), F.R.C.S. (Eng. and Ed.), Surgeon, Cumberland Infirmary, Carlisle. Crown 8vo. Pp. 476 + viii, with 246 illustrations. 1928. Edinburgh and London: Oliver & Boyd. 12s. 6d. net.

THE student of twenty-five or thirty years ago was very familiar with a little book on practical surgery by Caird and Cathcart. The work at present under review is a lineal descendant, and although it is a little larger it can still be carried in the pocket. It is a very useful book for students, containing chapters on "Case-taking", "Bandaging", "Wound Treatment", "Fractures", "Minor Operations", and many other details of the craft of surgery. There is an interesting addition, useful for men starting in practice, on "Hospital Economy", together with suggestions and plans by an architect for the erection of a simply constructed operating theatre.

Manual of Surgery (Rose and Carless). By ALBERT CARLESS, C.B.E., M.B., M.S. (Lond.), F.R.C.S., Emeritus Professor of Surgery, King's College, London; Consulting Surgeon, King's College Hospital, etc.; and CECIL P. G. WAKELEY, F.R.C.S. (Eng.), Erasmus Wilson Lecturer, Royal College of Surgeons of England; Assistant Surgeon, King's College Hospital, etc. Twelfth edition. Demy 8vo. Pp. 1544 + xii, with 639 illustrations in the text, 17 coloured plates, and 78 special plates of radiograms. 1927. London: Baillière, Tindall & Cox. 30s. net.

WE extend our most hearty congratulations to the authors of this well-known book. One of the original authors appears now to be spending his leisure time in travelling about to clinics overseas and in keeping himself abreast of current surgical teaching. The co-operation of one of the most active of the younger surgeons of King's College Hospital has been invoked in the production of this new edition. An eloquent tribute to the work and influence of Lister is given in the Preface. The alterations and additions in the text are especially in the direction of emphasizing the initial symptoms rather than the terminal phenomena of diseases, and laboratory methods of diagnosis are described more fully, while minute details of operations are given less space. We accord hearty commendation to the very full account which is given of fractures and their treatment, together with the special radiographic supplement. We rather doubt, however, the value of the chapter on military surgery, as it is too brief to be of any real use. A point on which the book seems to run a risk of losing its premier place among English text-books is that it is becoming too bulky, and we trust that the authors will see their way to cutting out some of the redundant material—such as the descriptions of alternative methods of treatment.

Nachbehandlung nach chirurgischen Eingriffen. By Professor Dr. EDUARD MELCHIOR, Oberarzt der Chirurgischen Universitätsklinik, Breslau. Royal 8vo. Pp. 339, with 32 illustrations. 1928. Leipzig: Johann Ambrosius Barth. Paper covers, M. 17.40; bound, M. 20.

THIS carefully written book is intended to be a guide for both the student and the practitioner in order to help them to understand the nature and treatment of the various complications and accidents which may arise after operations. A large part of the book is devoted to the consideration of general principles of disorder and treatment. Disturbances of the respiratory tract, the circulation, the stomach and intestines, are described systematically. First, loss of blood and difficulties of feeding are discussed in some detail. In considering the length of time that patients should remain in bed after operations, the author takes a position intermediate between the extremes advocated by various authorities. That is to say, whilst he advises that patients who have had operations on the head or chest should get up one or two days afterwards, yet he advises the retention in bed of hernia cases for at least ten days, and he further admits that getting patients up early does not prevent the possibility of pulmonary embolism. In discussing the treatment of the more important complications after operations the author offers a considerable number of alternative methods. Perhaps this is liable to leave the reader in some doubt as to which he should choose—for example, in the treatment of ileus the uses are described of various purgatives administered by injection into the circulation. Not only are the older remedies such as atropine, strychnine, and pituitrin mentioned, but also the active principles of senna and cascara under the names of sematin and peristaltin are put forward as reliable methods of treatment, and we

feel that this multiplicity of remedies is bewildering rather than helpful. Only five chapters are devoted to special operations as against thirty-two chapters descriptive of general principles. We think that the description of special operations and their after-treatment is of considerably less value than the first part of the book. It is curious to see what is almost identical with a Thomas's splint described and figured as Bruns's walking splint. As a book of reference in cases of difficulties and complications after operations we think this work will be of great value.

Lehrbuch der Chirurgie. By Professor Dr. C. GARRÈ, Bonn, and Professor Dr. A. BORCHARD, Berlin. Sixth edition. Royal 8vo. Pp. 752 + xxii, with 590 illustrations (some partly in colour). 1928. Leipzig: F. C. W. Vogel. Paper covers, M. 36; bound, M. 40.

THIS well-known German text-book of surgery was first published in 1920, and the fact that it has already reached a sixth edition shows that it has found ready acceptance among those for whom it was written. It represents an attempt to solve the very difficult problem of giving a short and clear exposition of surgical principles without encumbering the matter with many technical details. When this attempt is made by two such well-known teachers as Garrè and Borchard it at once commands our attention. The present edition has a special and pathetic interest because of the fact that its senior author died just after he had completed his contribution.

After a short and stimulating chapter on the teaching and learning of surgery and still shorter sections on the arrest of hæmorrhage, the treatment of wounds, and anæsthesia, the great bulk of the work is devoted to a description of regional surgery. In presenting an account of the injuries and diseases of each part of the body only those subjects are taken in any detail which are of common occurrence and of great practical importance. The selection and execution of the illustrations have been carried out with great judgement. At the end of the book is a short synopsis of the different surgical regions of the body with a special view to aiding diagnosis; this summary is, however, almost too compressed to be of much practical value. The last chapter deals with operative surgery, and is evidently designed for the purpose of conducting a course on the dead body. The ligation of arteries and the old classical but now obsolete amputations are described in a detail which they do not deserve. The most notable omission from the book is the absence of any reference to general surgical diseases, such as septicæmia, tetany, and syphilis, and in a book which mainly deals with general principles we regard this omission as important.

Operative Surgery. By J. SHELTON HORSLEY, M.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va. Third edition. Royal 8vo. Pp. 893, with 756 illustrations. 1928. London: Henry Kimpton. 63s. net.

A USEFUL short chapter on general considerations is followed by another on surgical drainage, which is regarded as helping nature's effort to extrude a foreign substance by reversing the flow of lymph. There is a somewhat sketchy chapter on operative technique. Eight small figures and half a page of description are given to Grant's method of tying a reef knot with forceps, which is excessive.

Chapter 7 deals with the suturing of blood-vessels. The author's ingenious and simple spring staff for triangulating the ends of the vessel makes the operation look easy, and laboratory workers should note it. In surgical practice the occasions when suture is preferable to ligation must be very rare. The indications are clearly set forth and the descriptions are excellent. Peri-arterial sympathectomy is also discussed. We have so much to learn about the balance of the arterial and venous circulations that the author is wisely non-committal. Early senile gangrene, which is omitted, is certainly more an indication for it than is a painful amputation stump. This chapter is one of the best in the book, and is followed by a short one on reversal of the circulation in which the experimental findings with the fallacies of the procedure are well described. Twenty-seven pages follow on the ligation of blood-vessels, the selection being judicious. There is no mention of the stay-knot of

Ballance and Edmunds. Few of us have known of a ligature slipping off a divided main vessel. Two ligatures are recommended for the proximal end. The reviewer has sometimes placed a transfixion ligature beyond his first one from apprehension, but a muscle-flap is better. Chapter 10 on aneurysm and Chapter 11 on arterio-venous aneurysm are both masterly. In all the part of the book dealing with the blood-vessels the experimental work done by the author and his son has clearly influenced the excellent presentment of the subject.

Chapter 14, a new one, discusses the causes of cicatricial contracture and forms a fitting introduction to Chapter 15 on plastic surgery. The remarks on venous stasis as a cause of the death of a pedicle-flap are particularly valuable. This chapter as a whole is clear and complete: some of the descriptions have evidently been rewritten from Davis's work, which is freely quoted. We confess to having had no need to use the Catlin knife and elevate the epidermis in cutting a Thiersch graft: there is no one method, and much depends on the sharpness of the razor and the contour of the thigh.

Chapter 17 is a well-balanced one on the operative procedures on the skull and brain. Souttar's craniectomy should be mentioned and its use figured in a future edition. The subject of anæsthesia also requires consideration here.

Chapter 22 deals with the thorax. From the description of a case of cardiomyolysis, the impression is received that the sole object is to free the pericardium from the chest. An enlarged heart hypertrophies, and no doubt freeing the heart from the pericardium is also advisable in some cases. The excellent results when the bony restraint is removed are not mentioned. Sufficient emphasis is not given to the more usual combination of decortication of the lung with the Estlander operation. In the reviewer's experience the lung if sufficiently freed is never too old to expand if its substance has not been the site of abscess. The subject of abscess of the lung should be introduced by pointing out that at least three months upside-down drainage six times a day *per vias naturales* with respiratory disinfection should be carried out first; the drainage operation will then seldom be required. The descriptions of it and of lobectomy are good. Operations on the heart for wounds and mitral disease are given; the useful fact that a stab wound of a ventricle does not bleed during systole is not mentioned. Cutler's incision for valvulotomy is just where aneurysm of the ventricle usually arises in a syphilitic patient: Souttar's method seems on the whole to be of better promise. Phrenic avulsion and thoracoplasty are considered in their separate and joint aspects. A good chapter closes with a description of excision of the scapula and clavicle.

The author is evidently a great advocate of pyloroplasty, and his own method is well described. There is a redundancy of figures for gastro-enterostomy with and without clamps. "Inversion (of the stump of the appendix) produces a beginning intussusception and a protuberance on the mucosa of the cæcum which may possibly be the starting point for ulceration or for cancer". The facts have yet to be demonstrated. Bunt's diverticulum can often be seen at a second operation and appears to be of no clinical importance at all. Battle's incision should be described as well as McBurney's.

This book is essentially modern. There is one reference to the last century (1884) and one to 1891: most of the others are to post-war statistics. A short historical résumé of the evolution of any main operative procedure, with the reasons for the chief modifications and the late results of them should be a *bonum* in a book of this size, as it makes the study of the subjects so much easier. The work on the operative technique is frequently referred to as the "subject" which it is not. The work as a whole could be improved by the removal of certain parts, by omissions, and by additions, and the readability by using heavier type for heading paragraphs. Misprints are few, but a few are noticed being on pages 72, 697, and 866. The reference in the last line of page 1 is incorrectly given. Occasionally the diction needs amending, as "the subject" is highly important to lose no more blood than possible. The book is excellent and well reproduced. If they were headed and numbered the descriptions beneath would shorten the text very considerably.

of the insertion of more important matter. "The gloved fingers of the assistant" are conspicuously absent, and so are illustrations of instruments except where necessary. The index is excellent and renders the eighteen-page list of illustrations superfluous. The book evidently records experience, notably in the accounts of some of the laboratory work. It should be of value to the profession.

A **Text-book of Surgical Diagnosis.** Edited by A. J. WALTON, M.S., F.R.C.S., B.Sc., M.B., Surgeon, London Hospital; Late Hunterian Professor, R.C.S., England. In two volumes. Medium 8vo. Pp. 1121 + xi, with 570 illustrations. 1928. London: Edward Arnold & Co. 63s. net.

THE work is edited by one occupying an eminent position in the profession. In his preface the editor says, "the weakness of modern surgery lies in diagnosis", and offers these volumes as a help towards the remedy. He has contrived, as he claims, to have brought together the contributions of no fewer than fifty-two men, almost all of whom have unusual knowledge and experience in their subject. Five of his contributors are from the provinces, and one (Dr. Allen B. Kanavel) writes from Chicago the chapter on "Acute Infections of the Hand"—a subject in which he is the acknowledged expert.

The book covers the whole range of surgery; it includes articles on "Suppuration and Inflammation" (Sir George Lenthal Cheatle), on "Shock, Collapse, Delirium, Hæmorrhage and Hæmophilia" (Russell Howard), "Specific Infections" at home and abroad, very widely spread articles on orthopædic subjects, and chapters on such special branches as the "Pituitary Gland", "Thyroid and Parathyroids", "Thymus and Carotid Bodies" (by the editor). The "Female Genital Tract" and "Genito-urinary Surgery" are adequately dealt with by well-known authorities. The "Nose and Pharynx", the "Larynx and Air-passages", and the "Ear" are very carefully written (Norman Patterson). Mr. P. N. Panton contributes an all-too-short chapter on "Diagnostic Use of Laboratory Methods". Dr. Sequeira and Mr. David Lees, of Edinburgh, write respectively on "Syphilis" and "Gonorrhœa".

The editor hopes the book will be of "real service to the busy practitioner", and doubtless there are many grounds for such a pious hope, but at the moment of review one happened to have an obscure case of jaundice under one's care—a by no means uncommon type of case. Acting as a 'busy practitioner', one hoped to solve the problem by reference to the index in this book, only to be met with two references, one to 'hæmolytic jaundice', the other to 'acute pancreatitis'. Such a book might well have discussed the diagnosis of jaundice in a separate chapter, with all the tests, symptoms, and signs put down succinctly. It would be impossible to offer serious criticism of any individual article; they are uniformly excellent. The very nature of the book makes the articles departmental and not coherent with one another. Some are too full and others too short—no different result could have been arrived at with so numerous and diverse a body of contributors.

For the surgeon and the student the book is eminently desirable, and it is a great contribution to surgical literature. Such serious criticism as can be made—and it is serious—is in the choice and reproduction of many of the illustrations, particularly the radiograms. They are not worthy of the book. Many of the photographs only feebly show what is intended, some do not show the intention at all.

Die Chirurgie des Land-, Schiffs-, und Kolonial Arztes. By Professor W. VON OETTINGEN. Royal 8vo. Pp. 388 + xx. 1928. Dresden and Leipzig: Theodor Steinkopff. Paper covers, M. 18; bound M. 40.

THE author of this book, who has been working for the last five years in South America, has been impressed with the need for providing a short synopsis of surgery for the needs of those busy country practitioners or ship's doctors who cannot be burdened with large text-books and who have no access to reference libraries. There can be no doubt that he has succeeded in condensing an enormous amount of useful information into a small compass, but the question must arise whether

information in such condensed form will be of much value to those who are really seeking enlightenment—for example, all the injuries of the lower limb, including fractures and dislocations, are described in ten pages. In regard to treatment, the absence of any illustrations or diagrams is a great drawback. The book concludes with a short list of what is regarded as the best equipment in the way of instruments, apparatus, and drugs for the country doctor.

Surgical Nursing and After-treatment: A Handbook for Nurses and Others. By H. C. RUTHERFORD DARLING, M.D., M.S. (Lond.), F.R.C.S. (Eng.), F.R.F.P.S. (Glasgow), Surgeon to the Coast Hospital and Lidcombe State Hospital, Sydney, N.S.W. Third edition. Pp. 626, with 149 illustrations. 1928. London: J. & A. Churchill. 8s. 6d. net.

It is only right and proper that every large school of training for nurses should have its own text-book of instruction written by the teachers of the school. The present volume represents an Australian text-book for the nurses of Sydney. The fact that it has already reached its third edition is sufficient to show that the book has been appreciated. It contains a very full account from a nurse's point of view of general surgical principles, operations, and after-treatment. It is divided into two sections, the first dealing with general surgical nursing and the second with regional surgical nursing. The book is well illustrated by many pictures of surgical appliances, pathological processes, and operations. A very useful appendix, dealing with weights, measures, prescriptions, and nursing methods concludes the volume. The book represents a very high standard of nurses' training.

Diseases of the Larynx, including those of the Trachea, Large Bronchi, and Oesophagus. By HAROLD BARWELL, M.B. (Lond.), F.R.C.S. (Eng.), President of the Section of Laryngology, Royal Society of Medicine, etc. Third edition. Demy 8vo. Pp. 278 + xv, illustrated. 1928. London: Humphrey Milford. 12s. 6d. net.

THE advance of laryngology since the second edition of this book was published in 1910 has necessitated very considerable additions, particularly in the direction of endoscopic methods and in regard to the treatment of malignant disease of the larynx. The work has always been somewhat unusual in confining itself to diseases of the larynx, but in the first edition the author was eminently successful in achieving his object of supplying a manual suitable for the general physician, surgeon, and student. The additions now made to bring the work up to date have reached the same high standard as the original work. The illustrations are many and excellent, and those drawn by the author from his own cases representing the mirror pictures of the larynx in various conditions should prove of particular value. The book can be cordially recommended as a small but very comprehensive and up-to-date treatise on diseases of the larynx.

Plastic Surgery of the Orbit. By J. EASTMAN SHEEHAN, M.D., F.A.C.S., Professor of Plastic Surgery, New York Post-graduate Medical School and Hospital, etc.; with a Preface by PIERRE SEBILEAU, Professeur de la Faculté de Médecine de Paris. Imperial 8vo. Pp. 348 + xxi, illustrated. 1928. New York: The Macmillan Co. 50s. net.

THIS book is the second of the author's important contributions to the rapidly growing literature of plastic surgery. In it he has adhered to his evident conviction that it is wiser to write in great detail about a single branch of the subject than to skip incompletely over all its branches. To some extent the title of the book belies its contents, for there is included much material of general plastic interest to make up, as it seems, for the limited scope of the plastic work possible in the region under consideration.

The chief appeal of the book will be to the eye surgeon who wishes to extend his knowledge of plastic procedures in general and of their application to his field of activity. The student or graduate desiring to take up plastic work as a specialty will hesitate to purchase a volume of this size and price for instruction in so small

a branch, while the already-trained plastic surgeon will not wish to read through much with which he is already conversant before reaching the descriptions of operations in Part 3. The seventy-odd pages devoted to the anatomy and physiology of the skin, the preparation and after-care of patients, skin-grafts, and general surgical considerations are, however, very ably written and contain many original ideas. The reference to "what might appear at first glance to be over-elaboration of some of the earlier chapters" is perhaps necessary, for we cannot imagine that many patients would submit to the extensive pre-operative treatment recommended in them. Repeated steaming of the face skin, removal of comedones, cleansing with carbon tetrachloride, and various forms of light treatment may represent precautions which will give a slightly increased chance of successful results, but we feel that their application would prove impracticable in 90 per cent of cases. The disfigured patient has usually taken a long time to produce sufficient courage to seek treatment and, once having embarked upon the journey, is impatient to reach its end.

An interesting preface by Pierre Sebilcau, of Paris, is followed by an unusually long one by the author. The latter is particularly well written and amply justifies its length. In it is pointed out the unreasonableness of blaming unqualified persons who, without sufficient surgical knowledge, have tried to free people of those inferiority complexes which are constantly associated with disfigurement and deformity, unless we are prepared to substitute skilled qualified assistance and unless we spread knowledge of the possibilities of modern reconstructive surgery amongst members of the general medical profession so that sufferers may be guided to competent sources of treatment. With this is intimately connected the need for realizing that functional restoration rather than cosmetic improvement is the most important concern of the true plastic surgeon.

Patience, capacity for unlimited care over detail, and artistic aptitude are rightly stressed as necessary qualifications in the surgeon. Paraffin injections are justifiably condemned and a special appendix on this subject is wisely included later. The keloid scar is aptly styled the 'sword of Damocles' of plastic surgery. Later in the book the use of hygroscopic threads and exposure to X rays are mentioned as useful forms of treatment. We are surprised to find no mention of radium treatment in this connection.

Part 1 is chiefly concerned with the anatomy and physiology of the orbital region. Beautifully illustrated and clearly written, these pages serve a useful purpose in collecting together a fund of information which ordinarily would call for reference to a number of separate text-books. In Plate VI the internal carotid artery is wrongly labelled 'external carotid'. Part 2 deals with various general considerations. Local anaesthesia is recommended, general anaesthesia being reserved for children and very nervous adults. For the latter the double-tube endotracheal method is advised, and it is interesting to find an appendix describing this method in detail and written by I. W. Magill, of London, who first introduced it.

The importance is stressed of clean incision at right angles to the skin surface and arranged when possible in accordance with Langer's lines; of delicate handling of all grafts and indeed all tissues; of careful haemostasis with a minimum of ligature material; of avoiding tension on skin edges; and of early removal of sutures and covering dressings. The use of chop-sticks for handling grafts is an innovation. Metal clips for skin suture are condemned, and rightly so. As indicating the thoroughness of this section, reference may be made to instructions about hard beds without pillows for several days after operation; soft food to save chewing; non-residue diet, pure vegetable aloid (calsalettes), lactic oats, and even a prescription for a mixture for stimulating the circulation and activating kidney elimination.

A full description is given of all forms of free grafts, and Ferris Smith's pressure bags are given well-deserved notice. The mucous-membrane graft, advocated for restoration of eyelid lining, is carefully described. That obtained from the middle turbinate is stated to be the best match for ocular conjunctiva. An outline of the processes by which vitality is established in grafts and flaps and of the various means of ensuring that these processes shall act to their fullest extent makes very interesting and instructive reading.

Part 3 gives full descriptions of operative procedures, all profusely illustrated by clearly drawn diagrams. There are very few photographic records of actual results. Transplantation of single hair follicles for the replacement of eyelashes sounds somewhat fantastic, but statistics are given of many successful cases by Krusius, whose special instrument is illustrated. A considerable amount of space is devoted to the very difficult correction of marginal notches, and emphasis is laid on the necessity for keeping the line of surface approximation away from that of the tarsal reunion. The history of the treatment of blepharoptosis is reviewed and new operations by de Baseovics are fully described. Dacryocystitis, a condition not usually treated by the plastic surgeon, is fully covered, and a very clear description is given of the Dupuy-Dutemps operation for anastomosing the tear duct to the nasal mucous membrane. Several well-illustrated pages are devoted to rotation flaps, and due reference is made to the work of Esser and Imre in this connection.

We think it a pity that eleven methods of treating ectropion should have been resuscitated, for the author admits that very few of them are applicable to the cases which come into the hands of the plastic surgeon. More emphasis laid on this point might reduce the number of mutilated eyelids produced by what may be described as meddlesome surgery which has failed to analyse the nature of the deformity. It is not understood what is meant by a 'hollow' mould of Stent in the description of the treatment of ectropion by Thiersch grafting.

In Fig. 175, illustrating the operation of canthotomy, the sutures appear to be running in the wrong direction, and in the legend to Fig. 176 'Cantrorraphy' is evidently a printer's error. The general arrangement of the book is good, but there is some avoidable repetition.

Die Körpereigene freie Fascienverpflanzung: ihre experimentellen Grundlagen und ihre praktische Anwendung. By Dr. ERNST KÖNIG, Privatdozent für Chirurgie; Asst. der Chirurgischen, Universitätsklinik, Königsberg i. Pr. With a Foreword by Professor Dr. M. KIRSCHNER. Royal 8vo. Pp. 302 + vi, with 118 illustrations and 2 coloured plates. 1928. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, M. 20; bound, M. 23.

THIS is a very useful monograph on a subject of great practical importance. It is clearly written and well illustrated, and comprises both experimental and practical work published by more than six hundred workers, references to whose articles appear in a classified list at the end of the book. As the author himself remarks, the various operations for which free fascia transplants have been used vary very much in their value. It is quite possible that in many cases simpler operations might be used to effect the same result; but on the other hand, a well illustrated book like the present is a most valuable work of reference for those engaged in practical surgery. It is no doubt chiefly in connection with the injuries and paralysis of the limbs that fascia transplantation finds its most useful indication, but in the repair of cranial and abdominal defects also it is probably superior to any other method. There are two points in which we think that this very practical book might be improved. One is the consideration of the method of cutting out the fascial strips; there are now various mechanical devices by which a long strip of fascia can be removed without an extensive incision, but these are not referred to. The other point is a discussion of the ultimate fate of the transplanted fascia, and particularly whether its structure and elasticity are preserved.

The Essentials of Otolology. By GEORGE BIRMINGHAM McAULIFFE, A.B., M.D., F.A.C.S., Assistant Professor of Otolology, Cornell Medical College; Oculist and Aurist, Misi-cordia Hospital, New York. Demy 8vo. Pp. 117 + xv, illustrated. 1928. London: Humphrey Milford. 16s. net.

IN the opening words of the preface, "this book is the embodiment of the method of teaching that the author has used for twenty-five years". It is intended as a practical and easily understood presentation for the student, and from the point of view of the author's students, it will no doubt serve a useful purpose in supplying

them with a permanent record of his teaching; but for the student in general its value is more doubtful. The reviewer has found it very difficult to read with understanding, chiefly owing to what seems to be the unsystematic way in which the work is set out. An attempt is made to describe practical details of examination which can only be taught on the patient. In a small work designed for the junior student as many as sixteen illustrations should not have been devoted to the description of one particular operation. The reviewer feels that the work should be retained for use in the clinic in which it has originated.

The Mechanics of the Digestive Tract: An Introduction to Gastroenterology. By WALTER C. ALVAREZ, M.D., Associate Professor of Medicine, University of Minnesota (The Mayo Foundation). Second edition. Medium 8vo. Pp. 447 + xix, with 100 illustrations. 1928. London: William Heinemann (Medical Books) Ltd. 31s. 6d. net.

THE first edition of this book, published in 1921, set out to describe and to prove the theory that the movement of the contents of the digestive tract depended upon what the author called a 'rhythmic gradient'. Such a gradient was apparently actuated by metabolic processes and was independent of nerve influence. This is the second edition of the book, and it is practically new in that in expanding the theory it enters into and discusses the mechanics of the digestive tract as influenced both in health and disease by what may be called extrinsic action.

After describing the 'underlying metabolic gradient', the author discusses in detail the œsophagus, the stomach, the duodenum, and the small and large intestines. His account of the movements of the stomach is of great interest; he deals with the pain of ulcer, the effect on movements of the stomach of disease or of mutilation by operation, the *modus operandi* of a gastro-enterostomy, and devotes a chapter to the pylorus and duodenal cap.

It would be wrong to suppose that the book is useful only to the research worker, although it is true that the last chapter, which gives an account of technical methods and apparatus, is essentially so. One can imagine the ordinary operating surgeon keenly interested in almost every paragraph. Chapter 21 is a description of the mechanics of the gall-bladder, most attractively written and of exceeding interest. Chapter 25, on reverse peristalsis and its symptoms, must be of great utility to both surgeon and physician. Chapter 26, on flatulence, is a remarkable dissertation on this very trying condition and will well repay close attention. The book teems with statements which bring the reader to a sudden stop amazed at contradictions of many of the things taught him as a student—for example, it comes as a shock to the surgeon to find that the giving of milk increases the rate of progress of food residues through the small bowel.

The illustrations are remarkably well reproduced. The photographs of distinguished physiologists and other research workers are particularly welcome and interesting. The quality of the paper and of the printing is unimpeachable. Taken in all, this book is one of the most interesting we have had the pleasure of reading, and whilst one cannot by any means agree with the views expressed by the author or with the deductions drawn from the experiments, one cannot help being fascinated by his ingenuity.

The Injection Treatment of Varicose Veins. By A. H. DOUTHWAITE, M.D., M.R.C.P. (Lond.), Assistant Physician to Guy's Hospital. Third edition. Crown 8vo. Pp. 51 + x. 1928. London: H. K. Lewis & Co. Ltd. 4s. net.

THE success of this small book, which has justified the publication of a third edition, is the best evidence of its usefulness. It presents a careful account of what is now recognized as a simple, safe, and useful method of treating varicose veins. It is based upon an experience of nearly 2000 cases, and the author therefore considers himself justified in describing the essential procedure as perfectly simple, easy of performance, invariably effective, and quite safe if employed with reasonable care. He claims that the worst cases of varicose veins are those in which the most brilliant results are obtained. In the new edition the main changes relate to alternative solutions which the author has found useful. In the majority of cases he uses

quinine and urethane, and he now suggests that the following solutions are sometimes useful—namely, a mixture of sodium salicylate and sodium chloride, sodium chloride alone, or a 66 per cent solution of glucose. He does not support the suggestion of ligaturing the internal saphenous vein at the same time that the injection is made.

Etude chirurgicale des Gangrènes juveniles par Artérites chroniques non-syphilitiques. By RAYMOND LEIBOVICI, Ancien Interne des Hôpitaux de Paris. Medium 8vo. Pp. 230, with 20 illustrations. 1928. Paris: Gaston Doin et Cie. Fr. 25.

This monograph summarizes in a very convenient form our present knowledge of pre-senile gangrene, its pathology and treatment. The pathological appearances of the peripheral vessels and nerves are described and figured by microphotographs. The author attempts to classify pre-senile arteritis—following Moulouguet—into Buerger's disease and pre-senile arteriosclerosis, and he believes that discrimination is only possible in early stages. The chief etiological features are discussed—the susceptibility of Jews, and the influence of tobacco, cold, and infection. The mechanism of production of gangrene is dealt with and the paramount influence of obliteration of distal vessels demonstrated. Experiments on venous ligation are reported and its transitory result is shown by tracings. The symptomatology and hamatology are discussed and the clinical forms of arterial disease in the young set forth.

The estimation of the vitality of the limb is perhaps the most important section of the book. The author believes that Pachon's oscillography, while very valuable, is not entirely accurate. He considers lipiodol injection to be the most valuable means of estimating the vascular efficiency. An account is given of the differential diagnosis, and the medical methods of treatment are compared, with favourable emphasis on that of Silbert, who uses 5 per cent sodium chloride intravenous injections, and claims by their use a very gratifying avoidance of amputation so long as the patient abstains from smoking. The author's main thesis appears to be that amputations which are now performed for gangrene might safely be done more distally if the methods of investigating the vitality of the limb were used.

The book is thorough and impartial, and should be read by all who are interested in the subject.

Endokrinologie. Edited by Professor Dr. LEON ASHER (Berne), Professor Dr. ARTHUR BIEDL (Prague), and Professor Dr. HANS GÜNTHER (Leipzig). Vol. I, Part 1. Royal 8vo. Pp. 80, with 15 illustrations in the text and 2 plates. 1928. Leipzig: Johann Ambrosius Barth. M. 36 per volume.

This marks the beginning of yet one more review system dealing with medical subjects. It consists of twelve monthly parts, each of which contains two or three original articles in the nature of critical reviews, and then follows a series of abstracts from current literature relating to the problems of the endocrine glands.

The scope of the articles ranges over biological problems in the animal and plant kingdoms as well as those of the human being. The first number contains articles by de Quervain on infantile myxœdema; by Zondek upon the influence of the pituitary upon ovulation during pregnancy; and by Kisch on the adrenal tissues of the torpedo fish. No doubt this new journal will prove of very great value to research workers.

Surface Anatomy. By ARTHUR ROBINSON, M.D., F.R.C.S., Professor of Anatomy in the University of Edinburgh; and E. B. JAMIESON, M.D., Lecturer on Anatomy in the University of Edinburgh. Royal 8vo. Pp. 175 + vii, with 38 illustrations, of which 24 are coloured. 1928. London: Humphrey Milford. 15s. net.

This supplies an accurate and lucid account of the relations of the different tissues of the body to the surface, and fortunately for its value to the student it contains a great deal more. If the ultimate aim of the student learning anatomy is the study

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of medicine and surgery, writers of text-books of anatomy must have that viewpoint always before them. It is doubtful whether this book will appeal widely to men revising their anatomy for surgical examinations, because, being written by two 'pure' anatomists, it has none of that clinical insight which such readers very naturally seek.

Traité d'Urologie. By G. MARION, Professeur agrégé à la Faculté de Médecine de Paris; Chirurgien du Service Civile (Hôpital Lariboisière). Second edition. Royal 8vo. In two volumes. Pp. 1192, with 482 illustrations and 31 coloured plates. 1928. Paris: Masson et Cie. Fr. 200.

THE first edition of Professor Marion's book was reviewed in this JOURNAL in July, 1922. In his preface to the second edition, which has been entirely recast, the author points out that certain chapters have been added in order to incorporate in the work recent advances in urology; they are on the subjects of cystography, pyelography, cystoscopic appearances, renal infarct, the intercureteral bar, congenital hypertrophy of the neck of the bladder, the surgery of the urinary tract in diabetics, and vesical biopsies.

These two handsome volumes more than maintain the standard of excellence of their predecessors; the illustrations are beautifully reproduced, and, as was pointed out in the review of the first edition, the work is well worth buying for these without the text; we consider the microphotographs and the anatomical pictures to be the best we have seen. At the end of the second volume are placed a list of drugs which the author considers useful in genito-urinary surgery and a list of diets. The section on the technique of both the major and minor operations follows. We feel doubtful of the advantage of this segregation, and should prefer to have seen this part of the work scattered through the two volumes in the appropriate sections. We are sure that this book will retain its place amongst the standard treatises on genito-urinary surgery.

Röntgenology. By Professor ALBAN KÖHLER, Wiesbaden, Ex-president of the German Röntgen Society, etc. Translated from the fifth German edition by ARTHUR TURNBULL. Royal 8vo. Pp. 556 + xviii, with 324 illustrations. 1928. London: Baillière, Tindall & Cox. 42s. net.

THIS book is proclaimed to be of outstanding merit by the eminence of its author, by the rapidity with which it has reached the fifth edition in the original language, and by the very enthusiastic way in which it is introduced to our notice in its English guise by the various eminent people who join in the Preface. We never remember, by the way, having seen a book before which has five Prefaces and a Foreword.

The work deals with borderline conditions between mere anatomical variations and actual disease; it makes no attempt to describe or picture the ultimate or completed conditions of disease. Everyone familiar with the difficulty of X-ray diagnosis must welcome this most useful aid to their problems. Nearly three hundred pages are concerned with the description of the skeleton and the bones and joints. Of this part, that which deals with the hand and foot contains the greatest amount of information and detail, and we consider that the illustrations of the variations which occur in growth and development of these bones alone make the book well worth possessing. The author's name is generally known in this country in connection with that curious mal-development of the tarsal scaphoid concerning which there is still much doubt. The normal order of ossification, its common varieties, and the many abnormal bones which may be discovered in the hand or the foot, are all very carefully described. The latter half of the book is occupied with a description of the contents of the thorax and abdomen. The variations in the form and position of the heart are given in great detail and illustrated by a number of outline sketches. Pyelography and cystography are given only a passing reference.

The manner in which the book is illustrated is notable. The great majority of the illustrations take the form of line drawings. We very heartily commend

this feature, because X-ray pictures cannot be presented so as to show detail without viewing the negative by transillumination. It is much better therefore to give a clear diagram than a confused reproduction of a radiogram: the four illustrations (Fig. 83) of possible fractures and displacements of the calcaneum are a very good example of this. There are, however, two very definite criticisms which we would make of the book. One is that the great majority of the illustrations have not any legend attached to them, and it is therefore necessary to search the text to find the explanation of each picture. The other is that in relation to important diseases such as tuberculous disease of the spine, sacro-iliac joint, or hip, there might well be a few typical illustrations of the earliest manifestation of the disease. For example, this disease of the hip-joint is dismissed in less than one page, although of course it is true that a great many of the other pages describe other conditions which have to be distinguished from tuberculous hip.

We have no hesitation in saying that the book is quite invaluable for purposes of reference, and we congratulate both the publisher and translator on having brought this work within the reach of English readers.

Collected Papers of the Mayo Clinic. Edited by Mrs. M. H. MELLISH and H. B. BURTON LOGIE, M.D. Vol. XIX. Medium 8vo. Pp. 1330 + xx, illustrated. 1928. London and Philadelphia: W. B. Saunders Co. 60s. net.

THE material at the disposal of the publication committee comprises nearly 400 papers—a considerable output for the personnel of the Mayo Clinic, which now amounts to about 180; of these 100 are reproduced in full and over 200 are only mentioned by title.

Donald C. Balfour writes on the technique of partial gastrectomy for carcinoma; in describing the methods available for the re-establishment of gastro-intestinal continuity he contrasts posterior trans-mesocolic end-to-side anastomosis with anastomosis anterior to the colon. From the care which is advised for the closure of the duodenal stump it is evident that this is the danger spot of the operation. C. H. Mayo and Claude F. Dixon contribute a paper on a new method of permanent colostomy in which a bridge of skin is carried through the pelvic mesocolon so that when the exposed loop of colon is divided the upper and lower orifices are widely separated.

The wealth of material at the disposal of the Clinic is shown by Verne C. Hunt's review of 995 suprapubic prostatectomies carried out in a period of five years. In comparing the complete operation with two-stage prostatectomy he prefers the former, which in his opinion is applicable to 75 per cent of patients. Alfred W. Adson writes of 300 cases of cervical ribs. He describes an anterior approach which in his opinion has advantages over other methods.

Every thinking surgeon must realize how dependent surgical progress is upon a sound knowledge of the basic medical sciences. Dr. William Mayo's papers in this relation have been full of interest and information, and his contribution as Donald Church Balfour Lecturer to the University of Toronto is worthy of the subject and of the lecturer himself.

St. Bartholomew's Hospital Reports. Edited by W. McADAM ECCLES, Sir THOMAS HORDER, Bart., K.C.V.O., RONALD G. CANT, W. LANGDON BROWN, W. GIRLING BALL, and GEORGE EVANS. Vol. LXI. Demy 8vo. Pp. 260 + xxv, illustrated. 1928. London: John Murray. 21s. net.

THIS volume contains seven papers on the chest, of which at least five are of surgical interest. Dr. Hewer's review of anaesthesia in thoracic operations shows how many thoracic conditions are being drawn into the surgical fold.

Mr. A. E. Roche writes of three cases of torsion of the spermatic cord: he suggests that inversion may be an attempt on the part of nature to permit descent of the testis even though the cord be short. He recognizes incomplete descent and

intratunical mobility with inversion as predisposing causes of torsion ; but it is not surprising that he is unable to suggest as exciting cause any agent which will meet with general acceptance.

Sir D'Arcy Power writes on Harvey, St. Bartholomew's Hospital, and many other things directly and indirectly connected with the institution. It is the last which provides the pleasurable light element in a paper which is full of historical information.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXVIII (Vol. VIII, Fourth Series), No. 2, April, 1928. Medium 8vo. Pp. 127-252. 1928. London: Lancet Ltd. 12s. 6d. net.

IN this number there is an interesting article entitled "A Statistical Enquiry into the Etiology, Symptoms, Signs and Results of Treatment in 166 Cases of Gastric and Duodenal Ulcer". This paper is taken from the records of cases which have been investigated and treated in the New Lodge Clinic. A very careful analysis has been made of these patients, and the record is interesting reading both for physicians and surgeons.

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EPOCH-MAKING BOOKS IN BRITISH SURGERY.

By SIR D'ARCY POWER, K.B.E., LONDON.

VIII. CHESELDEN'S ANATOMY.

WE are accustomed to think of William Cheselden as a great operating surgeon; he thought of himself and was thought of by his contemporaries as an anatomist. In this sense the appreciation was correct: he made his living as a surgeon, but he was constant throughout his life to anatomy. He says: "The study of Anatomy, as it leads to the knowledge of nature and the art of healing, needs not many tedious descriptions nor minute dissections; what is most worth knowing is soonest learned and least the subject of disputes; while describing and dividing the parts, more than the knowledge of their uses requires, perplexes the learner and makes the science dry and difficult. In describing the part," he adds, "I have pretty much neglected the minutiae in Anatomy; Nor have I been very particular about those things which cannot be understood without being seen, and being seen need little description; but have endeavoured to be more explicit about those which are of greatest use in Philosophy, Physic and Surgery. And I could wish the dividing and distinguishing of parts more usually done with more regard to these valuable ends." There is no doubt, therefore, that Cheselden was the first in England to write on surgical or, as it is now called, applied anatomy and thus began the long line of surgeons who based their knowledge on anatomy—a line which lasted until pathology—as different from morbid anatomy—gained the upper hand only so late as our own generation. Until the end of the last century every applicant for a post on the staff of a teaching hospital in London graduated through the dissecting-room; he now aspires through the pathological laboratory.

The first edition of "The Anatomy of the Human Body illustrated with twenty-three Copper Plates of the most considerable parts. All done from Life" appeared as a slender octavo volume of 271 pages in the year 1713. Cheselden being then twenty-five years of age. It proved an instant success: edition succeeded edition, until the thirteenth appeared in 1792, just forty

years after the death of the author. The popularity of the book was due in part to the simple language in which it was written and to the novel manner in which surgical cases were mingled with the drier anatomical facts; in part to its price ("Bound five shillings" is printed on the title page of the fourth edition, 1730); chiefly perhaps to the beauty and fidelity of the illustrations. When the plates of the first three editions were worn out, "having printed between three and four thousand, I have broke them to pieces, with intention to make a new set in which no expence shall be spared, and, as I have taken care to do justice to the buyers of the former editions by giving them leave to change their books at a low price; so I do intend, if I print another to act in the same manner." Three years later he fulfilled his promise by issuing to subscribers of four guineas the magnificent *Osteographia*, which only met with a moderate success. The plates were broken up when three hundred copies had been struck off, an additional hundred being reserved in case a Latin or French edition should be called for. Reduced copies of the plates were, however, used in the later editions of the octavo *Anatomy*.

Cheselden was a true artist and had a lively sense of humour. He posed each skeleton himself for the artist, with the result that the *Osteographia* contains such amusing figures as the one represented in *Plate I*. The drawings are also to scale and were made by means of a camera lucida as is seen in the frontispiece (*Plate II*), where Gerard Vandergucht is drawing; John Belchier is arranging the skeleton and Samuel Sharp is taking a note. Belchier and Sharp were the two apprentices of Cheselden, and both became distinguished.

The position between the old and the new teaching of anatomy is well illustrated by the earlier editions of Cheselden's *Anatomy*. The first three issues have as an Appendix "Syllabus sive Index humani corporis partium præcipuarum Anatomicus in xxxv Prælectiones distinctas." It gives in very bald Latin and in the approved manner of the schools the headings of the thirty-five lectures which Cheselden gave as his anatomical course. Abraham Chovet and Edward Nourse each issued a similar syllabus showing that they were private teachers independent of the United Company of Barbers and Surgeons. The three syllabuses are as dry as dust, but it should be remembered that from them, and especially from Cheselden's, came the *Hunterian* school and the surgery of the eighteenth century.

From the fourth edition onwards Cheselden published with his *Anatomy* "A short historical account of cutting for the Stone", in which he tells of "the first twenty-seven patients cut [by the lateral route] and I believe all are living. Indeed I had cut thirty-one who recovered before I had one died, having cut four more who recovered between the time the twenty-eighth was cut and the time he died. But I scorn to use any fallacious method of representing my success. Some of these being cut in the hospital and some privately, the truth of this account may be suspected by those who do not know me. I cannot take the liberty to mention the names of private patients therefore I will give a detail of those only which I cut this way in the hospital, where the first twenty-five recovered, to the truth of everyone of which I had above twenty witnesses, and I do believe these patients are all living at this time. Many of the children had the small-pox during their cure and some had the measles." From the tables he supplies Cheselden seems to have had

Plate 1



regular field-days in his operating-theatre; thus, on March 27, 1727, he cut three persons for the stone; on April 12, 1727, four patients; again three on May 15, and so on to the end of the list in July, 1730.

The following passages show how he combined surgery with anatomy; speaking of the changes in bone he says: "A soldier that from a shot in his left groin, had the head of the Os Femoris broke, part of which came away through the wound, upon which the limb wasted, and, he dying of an Anasarca about a year after, I found the Os Femoris wasted about an inch in length, but so much in its thickness that when they were both dried and sawed lengthwise through their middles, the emaciated bone weighed thirty grains less than half the weight of the other thigh bone. From the appearance of this man and the firm connection of all the bones with their Epiphyses, I am persuaded he must have done growing before he received this wound; therefore unless he was taken lame into the service, which cannot be supposed, this bone must have wasted about thus much in that time."

Of the superior maxilla he writes: "Part of the sides of the cavities that lie next the nose are only membranes which make the cavities like drums, perhaps to give a grave sound to the voice when we let part of it through the nose. I have seen an imposthumation [suppuration] from rotten teeth in one of these cavities, which has been cured by drawing some of the last grinding-teeth, and by making a perforation into it through their sockets. The drawing one or two of the last grinding-teeth generally, if not always, opens a passage into the Antrum, but if not or if the passage is not large enough, it may be made or enlarged with a carpenter's nail-piercer or gimblet which is as good an instrument as can be for the purpose."

The levator ani is described as two pairs of muscles. "Fistula's in Ano that are within this muscle generally run in the direction of the gut and may be laid open into the gut with great safety; but those fistulas or rather abscesses that are frequently formed on the outside of the sphincter and usually surround it, all but where this muscle is connected to the Penis, cannot be opened far into the gut without totally dividing the Sphincter which, Authors say, renders the Sphincter ever after incapable of retaining the excrement. One instance of this kind I have known, but Mr. Berbeck of York, an excellent Surgeon and particularly famous for this operation, has assured me that he has often been forced to divide the Sphincter, which has made the patients unable to hold their excrements during their cure, but, the wounds being healed, they have retained them as well as ever."

Pricking the fascia in the antecubital fossa was always looked upon as a very disastrous accident when the barber-surgeon or his boy 'breathed a vein' with a dirty lancet. Cheselden explains the reason. "A puncture of the tendinous expansion of the Biceps cubiti Flexor is supposed to be always attended with grievous pain and inflammation and has, if we have not mistaken the cause sometimes proved mortal; yet the best of surgeons has given us instances of large tendons being cut and stitched without any bad symptoms and I have often seen them ulcerated and mortified without any more sign of pain than in other parts; so that I cannot see what the great mischief of pricking the tendinous Fascia is owing to, unless its laying so much upon the stretch which may be wholly avoided by bending the elbow and turning

OSTEOGRAPHIA,
OR THE
ANATOMY
OF THE
BONES.



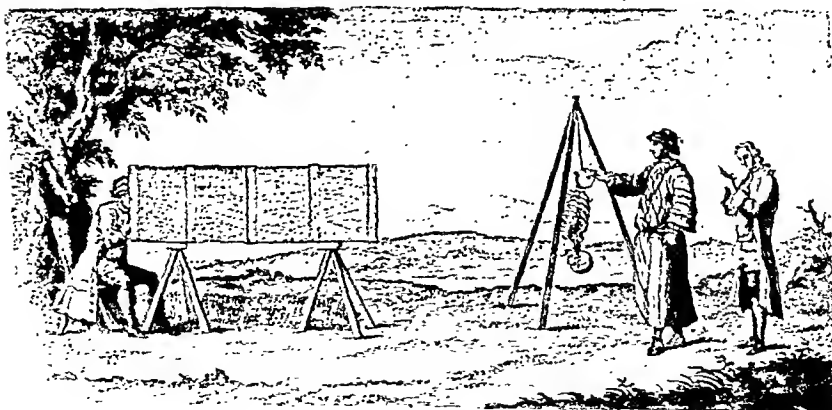
BY WILLIAM CHESELDEN

SURGEON TO HER MAJESTY;

F. R. S.

SURGEON TO ST THOMAS'S HOSPITAL,

AND MEMBER OF THE ROYAL ACADEMY OF SURGERY AT PARIS.



LONDON MDCCXXXIII.

the cubit prone. Since I have considered this ease, I have met with only one which was thus injured by an injudicious blood-letting who ordered the patient to keep her arm extended for fear of a contraction, and she was not without the most violent pain for a whole fortnight ; but upon bending the cubit and turning the arm prone, she grew presently easy and, in a few days, well. Nevertheless I am persuaded that most of the accidents which are thought to be merely from blood-letting are critical discharges of some Disease, and from the puncture a small inflammation beginning, increases and suppurates. But however singular I may be in this opinion, I can be sure I am disinterested in it having never had any ill accident follow blood-letting in my life."

"The best way of extirpating the tonsil is, I think, by ligature ; if the gland is small at its basis the ligature may be tied round it which I have easily performed by fixing the ligature to the end of a probe which I bent and so drew it round the gland and tied it. About five days after, the ligature growing loose I put on another in the same manner and then, in a few days, the gland dropped off. But meeting with another ease of this kind, where the basis of the gland was too large to tie, I contrived an instrument like a crooked needle set in a handle with an eye near the point. I thrust this instrument with a ligature in it through the bottom of the gland and then taking hold of the ligature with a hook I drew back the instrument ; then drawing the double ligature forwards I divided it and tied one part above and the other below in the same manner that I did to extirpate part of the Omentum in the cure of an Hernia."

And so the story goes on ; Cheselden teaches anatomy and surgery together in a manner wholly different from his predecessors.

Gerard Vandergueht, his artist, was born in 1696. He was elder son of Michiel Van Der Gueht, a Flemish engraver who came to England and was a pupil of David Loggan who engraved the well-known plates of the Colleges at Oxford and Cambridge. Gerard was employed by Sir Hans Sloane as well as by Cheselden, but he worked chiefly for the booksellers until he became a dealer and opened a 'Gallery' in Lower Brook Street. He died in 1776, his wife surviving him, the father of thirty or forty children, of whom Benjamin, the thirty-second, followed his father's profession.

**THROMBO-ANGIITIS OBLITERANS:
WITH SPECIAL REFERENCE TO A CASE INVOLVING
THE SPERMATIC VESSELS.**

BY A. LEE MCGREGOR,

ASSISTANT SURGEON AT THE TRANSVAAL MEMORIAL HOSPITAL FOR CHILDREN, JOHANNESBURG;

AND F. W. SIMSON,

PATHOLOGIST AT THE SOUTH AFRICAN INSTITUTE FOR MEDICAL RESEARCH, JOHANNESBURG.

INTRODUCTION.

IN 1908 Leo Buerger defined and named the condition of thrombo-angiitis obliterans; he rescued it from the lumber heap of gangrenes of unknown origin. His work is still the most authoritative on this entity. Buerger's disease remains to-day a subject of perennial interest because of its fatal character, the suffering and disability it causes in young people, the relative brutality which must be resorted to in the endeavour to stay its course, and the obscurity as to cause which envelops the affection. It is a disease which is now well known in those of its manifestations which affect the limbs; that it may affect other parts of the body is barely hinted at, and it is a matter of such profound importance that any light on this aspect of the condition must necessarily be put before the medical public as clearly as possible. This must be the excuse of the authors for publishing in detail the clinical and pathological aspects of a case of thrombo-angiitis of the spermatic vessels.

To make clear important clinical distinctions between the condition in the spermatic vessels and in the limbs, it is necessary first to review briefly the known facts of the disease in its typical peripheral distribution; subsequently the case will be dealt with fully.

Etiology.—Many theories have been put forward to explain the condition; syphilis has been suggested as a causative factor, but in a close study of thirty cases Buerger and Kaliski¹ showed that there is no connection between these two disorders.

Buerger's² view is as follows: "The disease is not an endarteritis obliterans; it is an occlusive thrombotic process involving the deep arteries and veins of the upper and lower extremities, or the superficial veins; the early stages of the disease manifest themselves in an inflammatory lesion which shows a specific and characteristic morphological picture while in the process of healing; and that in the early or acute stage purulent foci make their appearance which would suggest a microbial agent or infectious causative factor. No organism, however, has as yet been demonstrated, even in the superficial veins when these are in the stage of acute inflammation."

Rabinowitz,³ however, claims to have isolated a bacillus from the blood of the local lesion and from the general blood-stream. This organism has

been subcultured, and after inoculation has produced lesions in experimental animals similar in gross and microscopical pathology to those seen in the disease thrombo-angiitis obliterans.

Oppel⁴ considers the etiology from an entirely different aspect, namely, that the condition is due to hyperactivity of the suprarenal glands. He states that the theory is largely based on the fact that the blood serum of these patients contains a larger quantity of vasoconstrictor substances than the serum of healthy people. He elaborates the theory further by supposing that there is an excessive constrictor effect on the vasa vasorum followed by intimal degeneration and 'secondary thrombosis'. This theory is the basis of the operation of unilateral epinephrectomy which he performs for the disease.

Dmitrijew⁵ considers the cause to be hyperfunction of the suprarenal glands, and supports this by citing Duchinowa, who found an increase of vasoconstrictor substances in the blood six weeks after suprarenalectomy. Lettule, Marchak, and Boyer⁶ conclude on both clinical and histopathological grounds that the disease is an entity resembling leprosy, tuberculosis, and syphilis, due to some specific, but as yet undetermined, cause.

Only 3 of Buerger's 500 cases occurred in women, and only 4 of the 500 were not Jews. Telford and Stopford⁷ comment on the latter observation as follows: "The disease has in fact been called 'Die hebraische Krankheit'. This, however, can be no more than a topographical accident; each of our 4 cases was British." Tobacco smoking is held by Buerger and others to be an important predisposing factor, which probably acts by bringing about changes in the wall of the vessels, rendering them liable to attacks of inflammation and thrombosis. The onset is commonly in the third decade.

The great diversity of etiological factors which we have quoted indicates that there is still no settled or definite opinion as to the cause of this disease.

Distribution of the Lesions.—These affect one or both of the lower limbs in the vast majority of cases; in some the upper limbs are affected also. Regarding the participation of vessels other than those of the extremities, Buerger⁸ says little is known. He has noted the 'old type' of occlusion in the spermatic artery, and in a branch of the gastric artery; of profound interest, however, is a case in which he observed the typical 'acute' lesions in the spermatic vessels of the cord. In a young man who consulted him for a subacute enlargement of the testis, epididymis, and cord, the diagnosis of tuberculosis of the epididymis or thrombosis of the vessels of the cord was made. Orchidectomy yielded an interesting specimen in which most of the veins of the cord and tributaries about the testis were thrombosed. Microscopic examination revealed typical 'acute lesions' in many of the vessels.

In *Keene's Surgery* reference is made to a case in which Murphy observed the disease in the renal vessels.

Signs and Symptoms.—These have only been described as they affect the limb vessels. Clinically the first stage is often an acute phlebitis of the superficial veins. The arteries may escape for years. Some swelling and heaviness of the feet may occur, and often there are tender inflammatory nodules in and below the skin. Later there is, as a rule, complaint of pain

in the legs. The symptom of intermittent claudication is very usual. The pains, at first intermitting, later become continuous and excruciating. Pronounced vasomotor and trophic disturbances occur: erythromelia, ischaemia, cyanosis, ulcers, and gangrene. Such is very briefly the clinical aspect.

Treatment.—Mueller⁹ states that we have nothing to offer the patient which assures any reasonable promise of success, except amputation. Stradin¹⁰ and others report good results from Leriche's sympathectomy. Koyano¹¹ states that Ringer's solution given hypodermically makes amputation superfluous; the intravenous method is the most general (Silbert).¹² Stühlern¹³ reports good results with small doses of pilocarpine and insulin. Philips and Tunick¹⁴ claim remarkable results with X rays which they attribute to the effect of the irradiation on the paravertebral sympathetic ganglia, and to the alterations produced in the circulating cellular blood elements (phagocytosis). Oppel's epinephrectomy has been alluded to above.

CASE REPORT.

P. L., age 28, Russian Jew, produce buyer, moderate cigarette smoker, consulted one of the authors (Lee McG.) in private on Dec. 14, 1927. His complaint was of a dull nagging irritation over the penis, left half of scrotum, and lower abdominal wall. This recurred daily and lasted for two to three hours. It was not sufficiently severe to interfere with work.

HISTORY.—On Nov. 15, 1927, the left testis was a little swollen and very painful. His doctor confined him to bed for four days. The previous history regarded children's diseases only, and venereal disease was denied. The family history was irrelevant.

EXAMINATION.—The left testis was a trifle smaller than the right; the epididymis contained two nodules in the caput and one in the cauda. The cord was perhaps a trifle thick (note taken at first examination). Sensation in testis normal. Per rectum, prostate and vesicles were normal; urine and urinary history normal. Other systems normal. Cutaneous tuberculin test gave a positive finding. Wassermann negative.

A tentative diagnosis of tuberculous epididymitis was made, a suspensory bandage ordered, and the patient told to report in two months. When further examined on Feb. 12, 1928, discomfort was less in scrotum. The nodules were considerably bigger. The vas deferens was as thick as a lead pencil at the external ring and much less thick at the upper pole of the testis. This was noted. (Fig. 407.)

A confident diagnosis of tuberculous epididymitis was made and operation advised. A urologist confirmed both this and the suggested treatment.

OPERATION.—On Feb. 15, 1928, under general anaesthesia, the testis was

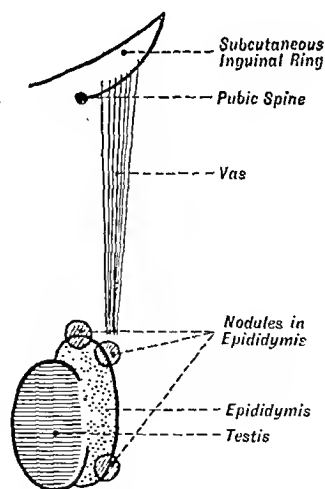


FIG. 407.—Diagram representing the condition found at the second clinical examination. Observe nodules in epididymis and thickening of upper part of vas.

removed with the eord as far as the internal ring. The wound healed by first intention, and the patient left hospital on the tenth day after operation.

The maeroscopic appearances of the specimen are dealt with later; here the only point requiring notice is the condition of the vas. On dissection the vas deferens was found to be normal. The apparent thickening of its upper part detected elinieally was due to the firm adhesion of many blood-vessels to the periphery of the vas, giving the impression on palpation that this tube was much thickened.

PATHOLOGICAL REPORT.—"Buerger's disease of the vessels of the eord." On receipt of this report a complete examination of the peripheral pulses revealed no abnormality.

POST-OPERATIVE PROGRESS.—For a week the patient had slight shooting pains up the right eord. These lasted perhaps five minutes at a time. They recurred daily. A nodule the size of a pin's head was felt in the eord near the testis. Vas normal. On March 13 the pains in the eord were much decreased, and on April 24 the patient said he still felt occasional discomfort in the right eord.

PATHOLOGY.

Since Buerger⁸ established beyond doubt that thrombo-angiitis obliterans is a definite entity and not to be confused with arteriosclerosis, many cases have been reported from various parts of the world. Notwithstanding the extent of the literature, comparatively few cases showing the acute lesions have been described. The rapidity with which the disease passes through the acute stage to the final healed process, the failure on the part of most patients to consult their medical advisers until the condition has become well advanced, and operation not being resorted to until more conservative treatment has failed to give relief, seem to explain satisfactorily the lack of material obtained for examination at this stage.

This difficulty of obtaining material in the acute stage has necessarily given rise to doubt whether thrombo-angiitis obliterans begins with an acute manifestation or not,* and the doubt will probably remain until many more cases showing purulent foci and giant-cell formation have been investigated. It is possible that the acute lesions may be found in conditions other than thrombo-angiitis obliterans, for Koyano¹¹ and Krampf¹⁶ claim to have found the same type of inflammation in acute thrombosis in infections. Be that as it may, the sequence of events leading from the initial acute change to the final stage of organization and canalization, so clearly shown by Buerger in both superficial and deep arteries and veins,¹⁷ suggests that, if not pathognomonic of thrombo-angiitis obliterans, it at least does occur in this disease. It is hoped that the histopathology of the present case which is described below will forge one more link in the chain of evidence in favour of this assumption.

* Perla¹⁵ states: "It is therefore of some significance that Buerger's so-called 'specific lesion' is never seen in the deeper arteries and veins, the *primary* seat of the disease. The lesions described in the migrating phlebitis are in all probability *not specific* for thrombo-angiitis obliterans."

Briefly, the changes found and described by Bucrger in thrombo-angiitis obliterans are, in chronological order: infiltration of the coats of the vessels and surrounding tissue by inflammatory cells, occlusive thrombosis of the affected part of the vessel, the formation of purulent foci in the clot, replacement of the leucocytic areas by altered angioblasts and giant cells, organization of the 'bland' portion of the clot *pari passu* with the same change in the giant-cell foci, and canalization of the organized tissue—the final product being organized and canalized vessels, associated with matting together of arteries, veins, and nerves by inflammatory fibrosis.

The spermatic vessels (*Case P.L.*) show all these changes; the veins are affected much more than the arteries, but the acute manifestation is confined to the veins. The spermatic vessels are slightly different in appearance and



FIG. 408.—Section of a vein stained by Weigert's elastic-tissue stain. The elastic tissue is more abundant than, but not so clearly defined as, that seen in the veins of equal calibre in the extremities.

distribution from those of the limbs. The arteries are small and few, the veins comparatively large, tortuous, and numerous; there is less supporting connective tissue, but elastic tissue is more abundant, both in the surrounding tissue and in the walls of the veins, while, in addition, it is less clearly defined than in other veins (*Fig. 408*).

The difference in the vascular distribution probably has no direct bearing on the lesion itself, but the large venous return compared with the small arterial supply probably accounts for the fact that the veins have suffered more than the arteries.

Many cases in which the vessels of the lower limbs were affected have been examined by one of us (F.W.S.), but in these the disease had lasted

many months or years and the histological picture showed only the chronic stage. In the case affecting the spermatie vessels, however, we have been more fortunate in that all stages of the disease are represented.

The maeroscopolical appearance of this specimen showed very little in the way of gross pathological change. There was slight general thickening of the cord and epididymis, somewhat more marked at the upper end of the cord. A few definite nodules were present at the upper and lower ends of the epididymis. These on section appeared to form part of the organ, were yellowish brown in colour, and quite unlike tuberculous aggregations. Transverse sections of the vessels of the cord and epididymis showed a varied type of occluding mass. In some vessels it was soft and red, in others firm, elastic, and white in colour. There was no definite abscess formation distinguishable

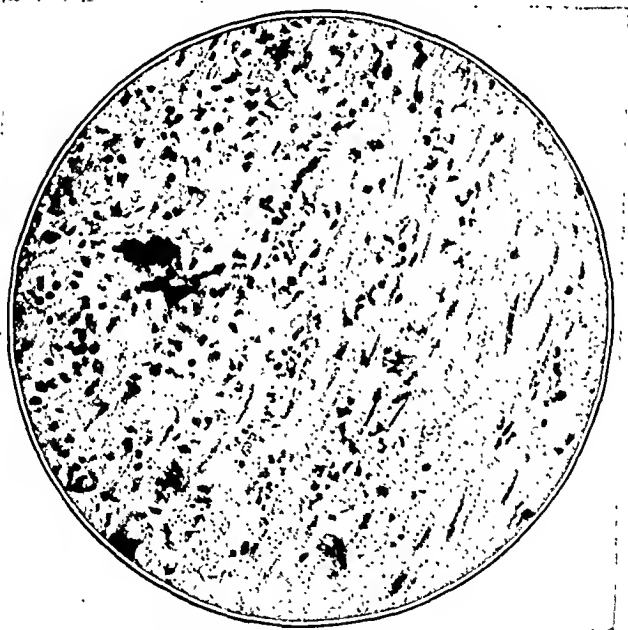


FIG. 409.—Showing the earliest acute manifestation. The muscle fibres of the media are seen separated by acute inflammatory cells, chiefly polymorphonuclears. The lumen of the vessel is above and to the left, where a giant cell may be seen in process of formation.

to the naked eye. The body of the testis showed no pathological change, and this was confirmed by subsequent histological examination.

Apart from the testis, sections were cut from many parts of the cord and epididymis, and these were taken, roughly, in serial manner in order to trace the change in type of inflammation in individual vessels. As the vessels in sections from the various parts were found to be affected in much the same way, a general description of the changes will suffice. For purposes of description, reference will be made to the microphotographs of the lesions in the affected spermatie vessels.

In Fig. 409 the disease is seen in its earliest manifestation. Besides the infiltration of the connective tissue surrounding the vessels by acute

inflammatory cells, the muscle fibres of the vascular wall are separated by the same type of inflammatory cells, as shown in the photograph. There are chiefly polymorphonuclear leucocytes, but a few large round mononuclears are also present.

In *Fig. 410* the lumen of a vessel is seen filled with soft clot. The clot is composed of fused platelets, fibrin, and red and white cells. The white cells are slightly increased in numbers, but not more so than could be explained by a general and local leucocytosis. Thomas¹⁸ has noted a persistent leucocytosis in early stages of the disease, so it is conceivable that a leucocytosis may show itself in the clot and be even more evident in the region of a local inflammatory process. As the disease progresses, miliary accumulations of polymorphonuclear leucocytes make their appearance at the periphery



FIG. 410.—A large vein completely filled with clot, the greater part of which is of the bland type. A few giant-cell foci are present at the periphery, and where these are absent organization by the simple method is taking place.

of the clot (Buerger's purulent foci) (*Fig. 411*). To form the foci, these cells can be seen wandering in from the vessels of the media. In some of the smaller collaterals the whole of the occluding thrombus may be replaced by the acute inflammatory cells. Passing to the next stage, such a focus as seen in *Fig. 412* shows almost total replacement of the polymorphonuclear leucocytes by altered angioblasts. The angioblasts attempt to penetrate the leucocytic area in order to form new capillary sprouts, having for their object the removal of the exudate as a preliminary to organization. The nature of the irritant in the leucocytic area prevents this, and the angioblasts become distorted and take on an irregular arrangement. Apparently some are able to enter and proliferate to form well-defined giant cells. The

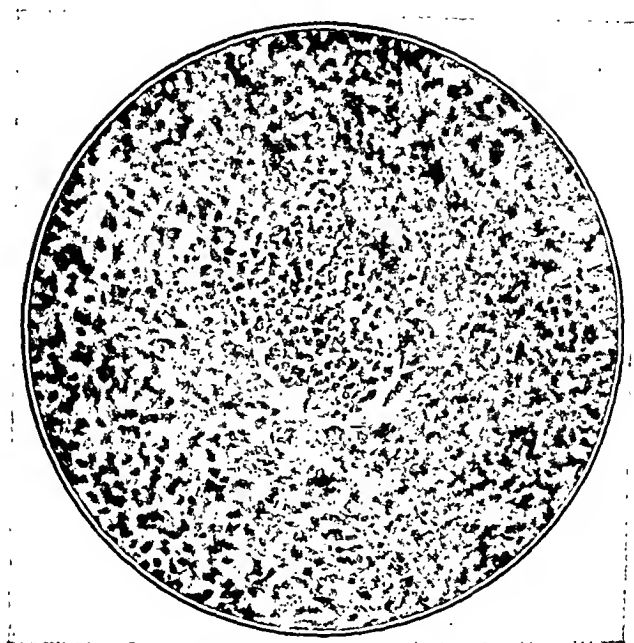


FIG. 411.—A 'purulent focus' in the peripheral part of the clot in a vein. Numerous polymorphonuclear cells are seen wandering towards this centre of infection.



FIG. 412.—Showing the peripheral part of the thrombus. Above and to the left is the vessel wall, to the right simple organizing tissue, and below a mass of fibrin. A giant-cell focus nearing completion is shown in the centre. The periphery of the focus is already replaced by angioblasts, the centre is occupied by a giant cell and a few remaining polymorphonuclear leucocytes.

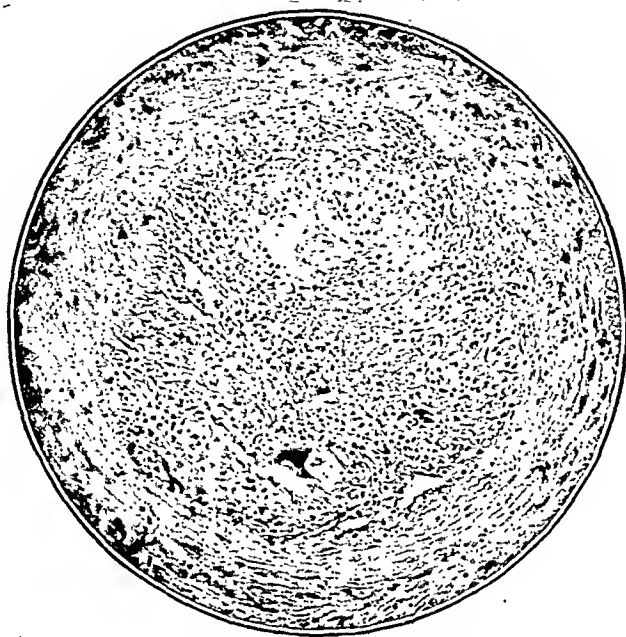


FIG. 413.—A vessel showing a small central zone of fibrinous clot. At the periphery several giant-cell foci may be seen, some of which have become partially organized.

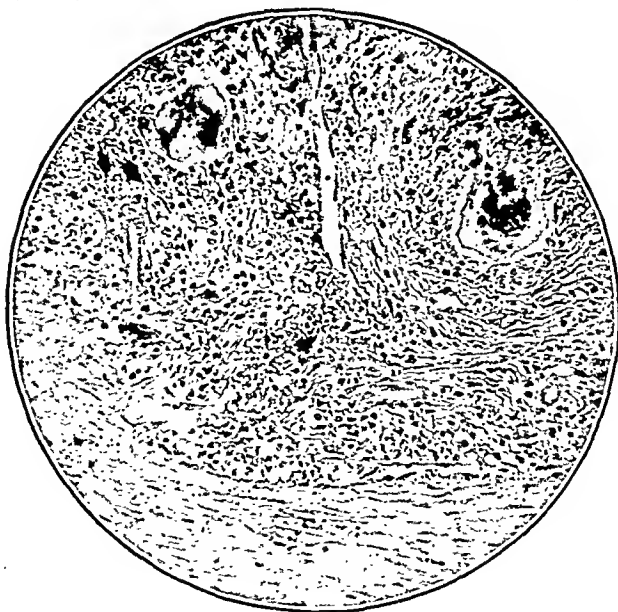


FIG. 414.—Two very large giant cells are shown, with numerous nuclei aggregated in the centre. The giant cells occupy the centre of partially organized foci.

giant cells often attain to a large size, are irregular in shape, and the numerous nuclei tend to form aggregations in the centre of the cell. When the leucocytes of the purulent foci have disappeared and angioblasts and giant cells have taken their place, the foci have a superficial resemblance to early tuberculous nodules, but a careful analysis will show how they differ. The central aggregation of nuclei is quite unlike the peripheral arrangement of the nuclei in the typical giant cell of syphilitic and tuberculous lesions; there is never any caseation; small mononuclear-celled infiltration at the periphery of a focus is absent, and, although carefully searched for, neither spirochaetes nor tubercle bacilli have as yet been found. The replacement of pus-cells by large mononuclear cells is evidently an intermediate stage in a special



FIG. 415.—Shows admirably the organization of the bland portion of the clot. On the left is the wall of the vessel from which new vessels and fibroblasts can be seen growing in to replace the constituents of uninfected thrombus. The darkly staining material is fibrin.

healing process, and this is not seen so definitely demonstrated in other forms of inflammation.

On completion of the giant-cell focus (*Figs. 413, 414*), capillary buds grow in from the vasa vasorum, accompanied by fibroblasts from the intima, and fibrous tissue is laid down. The giant cells are often last to disappear, and sometimes remain in evidence long after organization is complete in the remainder of the obturating mass. At the same time as the purulent foci are undergoing these changes, organization of the bland portion of the clot is proceeding by a simpler method. There is no intermediate replacement of the constituents of the clot by angioblasts, but young capillaries and fibroblasts grow in, the fibrin, platelets, etc., are absorbed, and the

whole mass is converted into young connective tissue (*Fig. 415*). The characteristic appearance of the occluding thrombus at the termination of this stage is a diffuse distribution of young capillaries lying amongst fibroblasts and small mononuclear and plasma cells (*Fig. 416*). In addition, in some vessels, there is a deposit of iron-containing pigment derived from liberated hæmoglobin from broken-down red corpuscles. The capillaries in different parts of the thrombus communicate, and in many vessels the continuity of the lumen is re-established, although only to a limited extent. With increasing dilatation and function, many of these new canalizing channels develop definite muscular coats and elastic tissue in the walls. Some of the arteries show thickening and increased corrugation of the internal elastic lamina,



FIG. 416.—Showing a part of a vein. The obturating mass shows diffuse distribution of young capillaries and early canalizing vessels lying amongst fibroblasts and small mononuclear and plasma cells.

but no reduplication or fragmentation worthy of note has been detected in the veins or arteries.

While the above description gives the sequence of events in this intermediate stage of organization, often in old-standing lesions the occluding mass is seen to be infiltrated by enormous numbers of cells; capillaries are few and connective-tissue corpuscles scanty, the picture presenting the appearance of chronic inflammatory granulation tissue.

Under normal conditions a vessel the seat of simple thrombosis may ultimately become converted into a fibrous-tissue cord, which represents the fibrosed wall enclosing the organized thrombus, but if infection is present in the thrombus, and if it continues, organization is indefinitely delayed. In

thrombo-angiitis obliterans, infection in a mild and chronic form is probably continued after the acute manifestation has subsided; this may account for the changes simulating chronic inflammatory granulation tissue in the older forms of the disease.

The acute inflammatory infiltration of the media of all vessels is soon superseded by cuffing of the new inflammatory vessels with cells of a chronic type, accompanied by a certain amount of fibrosis. A similar change occurs in the adventitia and surrounding supporting tissue, terminating in replacement of the normal fatty and loose connective tissue by inflammatory fibrosis, giving rise to matting together of arteries, veins, and nerves. (*Fig. 417.*)



FIG. 417.—This vessel shows the old or healed stage of the disease. The vessel is surrounded by inflammatory fibrosis. Many new vessels are present in the adventitia, and the lumen is occluded by a connective tissue, which is heavily infiltrated by small mononuclear and plasma cells. Numerous canalizing vessels may also be seen.

Fig. 418 typifies a change seen in some of the arterics. Below and to the right a canalizing sinus is seen eccentrically placed, crowding the organized clot into a crescentic mass. In the same artery, stained by Weigert's method to show elastic tissue (*Fig. 419*), points of difference between this lesion and those of atheroma and arteriosclerosis are brought out. In atheroma there is degeneration in the deeper parts of the thickened intima; this is absent in our case. In arteriosclerosis there is hypertrophy of the elastic tissue, with splitting off of new laminae from the internal elastic lamina, these laminae appearing in the new-formed tissue of the thickened intima; this is also absent in our case.

With regard to the histological diagnosis of these changes in the spermatoc vessels, there can be little doubt that we are dealing with a case of

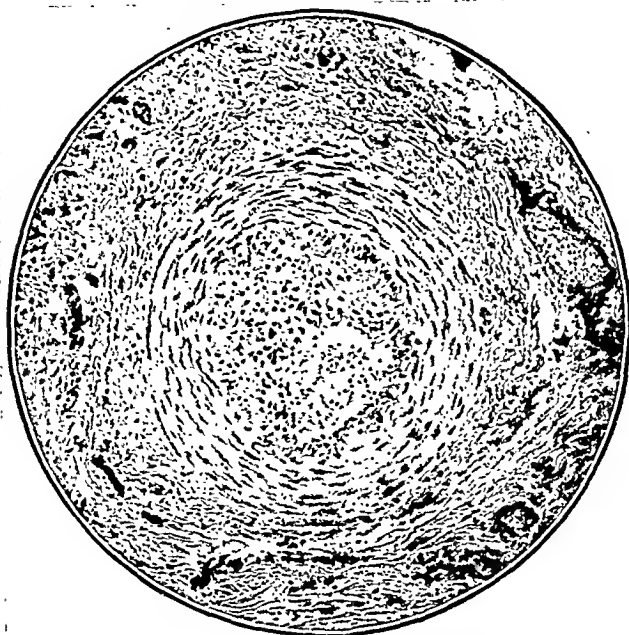


FIG. 418.—Section of an artery showing the inflammatory fibrosis surrounding the vessel. Numerous small mononuclear cells and new vessels may be seen in this tissue. The muscular coat is apparently healthy. The lumen is partially filled with new-formed connective tissue, and one large canalizing sinus is shown below and to the right. The new-formed connective tissue in the lumen is composed of cells arranged in an irregular manner. There is no evidence of degeneration or calcification.



FIG. 419.—This shows the same artery as in Fig. 418 stained by Weigert's elastic-tissue stain. There is no fragmentation of the elastic lamina. Elastic tissue has developed in the wall of the large canalizing sinus.

thrombo-angiitis obliterans, as it so clearly resembles, in all its phases, the disease as described by Buerger. It is true that the veins are chiefly affected, but the disease can be traced step by step in serial sections of these vessels, from the first acute manifestation to the final stage of healing. There is every reason to believe that the arteries have passed through the same succession of changes, since the healed product is the same in both arteries and veins. The differential diagnosis between thrombo-angiitis obliterans and granulomata, such as syphilis and tuberculosis, should present no difficulty. Apart from the absence of history of these diseases and a negative Wassermann reaction, as has been shown already, the histological picture reveals several points of dissimilarity. In addition, an interesting feature is shown in the case of thrombo-angiitis associated with syphilis reported by Smith and Wells Patterson,¹⁹ namely, giant-cell foci may be seen lying external to the internal elastic lamina, a distinguishing feature not yet seen in the lesions of thrombo-angiitis obliterans.

There yet remains the possibility of some specific bacterial infection other than tuberculosis or syphilis. Sections of the vessels from this case, treated by all the well-known bacterial stains, have revealed no organism of a specific nature. This is in agreement with other investigators; the only exception being Rabinowitz, whose work has already been mentioned above.

DISCUSSION OF THE CASE.

It is noteworthy that the patient was a Russian Jew, 28 years old, a cigarette smoker and non-luetic.

Clinical Diagnosis.—There existed in this case before operation 'no differential diagnosis' (to quote a surgeon at St. Bartholomew's Hospital). It was thought that no condition could mimic tuberculous epididymitis so closely as this did. Gonorrhœa could be excluded, also the nodules were extremely unlike those of gonorrhœa.

In view of the pathological finding the most outstanding features are two: (1) The subjective nagging pains—tuberculous epididymitis is usually painless; (2) The thickening at the *upper end* of the vas. This thickening excited interest at the time it was observed. In tuberculous epididymitis the thickening in the vas begins at the testicular end and travels up. In this case the vas approximated to normal below, but became thicker as it was traced up. This was found to be due to the thickened veins and thrombosed vessels so adherent to the vas as to be taken as part of it.

Because of the rarity of the condition and the entire lack of knowledge (except Buerger's case) of the clinical picture, this condition of the cord is of the very first importance.

Buerger mistook his case for tuberculous epididymitis, precisely as was done in this case. The mistake is so easily made because the condition apes tuberculosis (note positive von Pirquet) so closely that one does not consider a differential diagnosis. The nodules (thrombosed veins) are indistinguishable from tuberculous nodules. It seems to the writers that the only conditions likely to assist the distinction are: (1) The dull testicular pains distributed to the penis, affected side of the scrotum, and lower abdominal wall; and

(2) The characteristic thickening of the upper part of the vas. This, in association with nodules in the epididymis, would seem to be pathognomonic of thrombo-angiitis obliterans of the vessels of the spermatic cord.

Finally, and in repetition, the case presents itself as follows: A young man, 28 years of age, of Russian Jewish extraction, who smokes, complains of dull nagging hemi-serotal pains, radiating medially and upwards. He has one or more nodules which seem to be in the epididymis. These nodules are slightly tender and are exactly like tuberculous nodules, and the vas is much thicker above than below.

Prognosis.—The disease is characterized by relapses, so that the prognosis is always doubtful. The outlook, apart from operation, must be extremely bad as regards the testis. The main vessel to the testis is the spermatic or testicular; if this becomes occluded, the branch of the superior vesical which goes to the vas may vicariously supply the testis, but it is extremely doubtful if this latter vessel would escape the disease. The prognosis for the organ is therefore bad, and gangrene is a conceivable termination. The prognosis for life is good if the disease is localized to the scrotum.

As regards the other testis, from what is known of the disease of the limbs there is a strong possibility that it may become affected with the lesion sooner or later. It is unwise to tell the patient this. A brother or some member of his family should be warned. These subjects are frequently foreign Jews, with marked neurotic taint. Our own patient is already suspicious because of the pains he has had in his remaining testis since operation.

Treatment.—Perusal of the literature and prolonged observation of these cases of Buerger's disease leave the conviction that the vast majority ultimately require amputation of limbs. The other methods of treatment may and do relieve symptoms temporarily, but it seems to the writers that this result is entirely dependent upon the collateral circulation and the stage of vascular occlusion reached. The time, however, ultimately arrives when gangrene occurs and amputation must be resorted to for its relief.

In cases where the spermatic vessels are affected it would be interesting to try palliative measures first, such as the use of hypertonic salines, etc. Hemi-castration could always be resorted to as a last resource in cases where gangrene of the testis occurs.

SUMMARY.

1. The features of thrombo-angiitis obliterans affecting vessels other than those of the limbs are unknown.

2. A case is presented where the affected vessels were those of the spermatic cord.

3. The clinical appearances are almost exactly those of tuberculous epididymitis, but on analysis a few points arise which assist the differentiation. These are (a) local and radiating pains, and (b) a considerable apparent thickening of the *upper* part of the vas.

4. The histological changes found in the spermatic vessels of this case are those of thrombo-angiitis obliterans.

5. The whole sequence of events has been traced, step by step, from the first acute manifestation, through all intermediate stages, to the final healed product. These, in the order in which they occur, are: acute inflammatory infiltration of the coats of the vessels and surrounding supporting tissues, occlusive thrombosis, the formation of purulent foci in the peripheral part of the clot, replacement of the leucocytic areas by giant-cell foci, and, finally, organization and canalization of the obturating mass, associated with matting together of neighbouring arteries, veins, and nerves by inflammatory fibrosis.

6. The histological findings support the view, held by Buerger, that thrombo-angiitis obliterans begins in the form of an acute inflammation, the causal agent of which has as yet not been found.

REFERENCES.

- ¹ BUERGER and KALISKI, *Med. Record*, 1910, Oct. 15.
- ² BUERGER, *The Circulatory Disturbances of the Extremities*, 1924, 277. Philadelphia: W. B. Saunders & Co.
- ³ RABINOWITZ, *Surg. Gynecol. and Obst.*, 1923, xxxvii, 353.
- ⁴ OPPEL, quoted by Sachs, *Jour. Med. Assoc. S. Africa*, 1927, May 14, 215.
- ⁵ DMITRIJEV, *Zentralb. f. Chir.*, 1925, lii, May, 1081.
- ⁶ LETULLE, MARCHAK, and BOYER, *Presse méd.*, 1928, Feb., 193.
- ⁷ TELFORD and STOPFORD, *Brit. Med. Jour.*, 1924, ii, 1036.
- ⁸ BUERGER, *Loc. cit.*
- ⁹ MUELLER, *Surg. Clin. of N. America*, 1922, ii, 172.
- ¹⁰ STRADIN, *Deut. Zeits. f. Chir.*, 1926, cxciv, Jan., 338.
- ¹¹ KOYANO, *Acta Scholae med. Univ. imp. Kioto*, 1922, iv, 498.
- ¹² SILBERT, *Jour. Amer. Med. Assoc.*, 1926, June, 1759.
- ¹³ STÜHLERN et al., *Med. Klinik*, 1925, Nov., 1765.
- ¹⁴ PHILIPS and TUNICK, *Jour. Amer. Med. Assoc.*, 1925, May, 1469.
- ¹⁵ PERLA, *Surg. Gynecol. and Obst.*, 1925, xli, 21.
- ¹⁶ KRAMPF, *Deut. Zeits. f. Chir.*, 1922, clxxiv, 387.
- ¹⁷ BUERGER, *Loc. cit.*, pp. 334-337 (illus.).
- ¹⁸ THOMAS, H. M., *Amer. Jour. Med. Sci.*, 1923, Jan., 86.
- ¹⁹ SMITH and WELLS PATTERSON, *Brit. Med. Jour.*, 1925, i, 227.

THE DEVELOPMENT OF CYSTS IN CONNECTION WITH THE SEMILUNAR CARTILAGES.

By ROBERT OLLERENSHAW,

HON. SURGEON IN CHARGE OF THE ORTHOPÆDIC DEPARTMENT, SALFORD ROYAL HOSPITAL, MANCHESTER.

IN 1921 I described in the *BRITISH JOURNAL OF SURGERY*¹ three cases upon which I had operated because of persistent pain in the knee associated with a cystic change in the external semilunar cartilage. These were the first instances of the condition recorded in this country, although four cases had already been described in the German literature.²⁻⁵ Since 1921 I have met with eighteen additional cases in my own practice, and, in the light of this unique experience, I propose to review my original description of the condition and its pathology. A number of isolated cases have been reported in American and British journals, but as these are readily available to everyone, and as no fresh points have been brought out by them, they will not be discussed.

In my former paper I referred to the fact that the cysts were found in the external cartilage, and up to that time my own (three) cases and all those reported in Germany had been on that side of the joint (*Figs. 420, 421*). In



FIGS. 420, 421.—Case 20. Typical appearance of cyst of external cartilage.

(a) Lateral view. (b) Anterior view.

my present series four out of the eighteen have occurred in the internal cartilage, so that, although the lesion presents itself chiefly in the external cartilage (seventeen times out of twenty-one in my own cases), it may be found in the internal cartilage also. The pathology of the lesion has received

considerable attention, and a number of observers have challenged the statement that the cysts are lined by endothelial cells. Several other surgeons have found the lining as described and have confirmed my views. Many sections have been cut from my specimens, and, although we have not been able in every case to demonstrate the endothelium, we have no doubt at all that



FIG. 422.—Case 11. Low-power view of typical section, showing septa between cysts.

in very well with the usual account. by Goldzieher, who states: "the larger are formed by fibrous tissue. Their inner surface is lined by a layer of flat cells resembling endothelial cells and probably arising from the lymph vessels. The nuclei are rod-shaped and the cytoplasm is scanty. Some of these cysts are separated from each other by septa which consist of a few connective-tissue fibres covered on both sides by the endothelium described above. It seems fair to assume that the larger cysts arose from the vacuoles in the cartilage, and the formation of an endothelial lining, as well as that of a connective-tissue wall, are only secondary." Fig. 422 shows the septa separating the cysts.

Bristow⁸ records eleven cases of his own and states that thirty cases are on record. He has found, in some of his specimens, what appears to be an endothelial lining to the cysts, but refuses to class the lining cells as definitely endothelial. In the same publication McMurray⁹ makes the

in most specimens the lining is clearly present (Figs. 422–427). Professor J. Shaw Dunn, of the Pathological Department of Manchester University, allows me to say that he is in entire agreement with this statement.

Kleinberg⁶ has published a report of one case of cyst in the external cartilage. In his report he mentions a case of Allison and O'Connor,⁷ who recorded three cases, and gives their conclusion: that "it is our belief that these cysts represent the end-result of a degenerative process caused by an interference with the blood-supply of the cartilage in this region, the exciting cause of which is a non-lacerating injury." Kleinberg gives a description of his own case which falls

The specimen is then reported upon



FIG. 423.—Case 7. High-power view of cyst lining.

dogmatic statement that the lining wall of the cysts 'has been shown' to consist of compressed cartilage cells.

The condition is, without doubt, a primary lesion of the cartilage. In nearly all cases there is a more or less definite history of an injury. In my opinion, following this injury pre-existing small spaces in the cartilage gradually



FIG. 424.—Case 11.

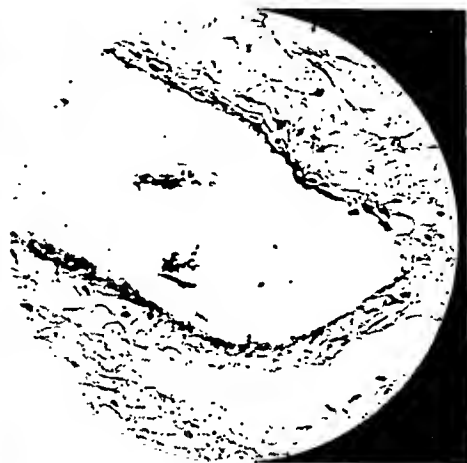


FIG. 425.—Case 4.

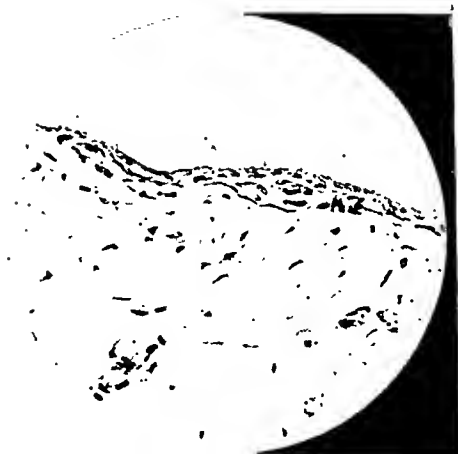


FIG. 426.—Case 20.



FIG. 427.—Case 15.

FIGS. 424-427.—High-power views of cyst lining.

enlarge, forming multiple cysts which are lined by an endothelial type of cell. In some sections, where no endothelium could be found, I believe that it had been detached in the preparation of the specimens. It is a very delicate layer, and, in cutting sections of a tissue containing material of such differing consistencies as fibrocartilage and the soft gelatinous content of the cystic

spaces, it is extremely easy to detach the lining cells. This is especially applicable to the larger cysts, where, I suspect, some have looked for the endothelial lining, neglecting the smaller spaces. In the sections which are here published there are, in my view, living cells which cannot possibly be classed as anything but endothelium (*Figs. 423-427*). They are certainly not in the least like connective-tissue cells flattened out under pressure. The view expressed by Goldzieher that the endothelium is derived from the lymphatics is an interesting one. In my original paper I advanced the opinion that the cysts were of developmental origin, and this is accepted by Zadek and Jaffe,¹⁰ who give a most detailed and excellent account of a specimen removed by them at the New York Hospital for Joint Diseases.

Their observations regarding the presence of a definite endothelium, particularly lining the smaller spaces, agree with my own.

Factors which favour the congenital theory are: (1) The multiplicity of the cysts; (2) The presence of papillary synovial inclusions without cyst formation (*Fig. 428*); (3) The absence of old or recent hæmorrhage into the cysts whether they are of old-standing or of recent development. The presence of a synovial endothelial lining in the smaller and medium-sized cysts refutes the idea that the cysts are ganglionic in character.

Kleinberg is, of course, wrong when he says that the condition "practically never invades the in-

ternal meniscus". My own series gives four cases out of twenty-one in the inner side, and Bristow's series gives two cases out of eleven—an almost exactly similar proportion. Working on this ratio, eighteen cases in one hundred should be in the internal cartilage.

Treatment.—In this connection I have nothing further to add to my paper published in 1921, in which it was stated that total removal of the cartilage with its cysts was essential for bringing about a cure. Bristow has pointed out the necessity of preserving the capsule of the joint when the dissection of the cartilage and cysts is being made. This is extremely important, and if it is improperly done, will prevent the proper approximation of the capsule.

None of the cases recorded below have given any trouble in their progress after operation, and all have recovered full function. In two of the older patients (*Cases 7, 19*) the osteo-arthritic changes present at operation appear to be stationary.

Figs. 429-432 show the macroscopic appearance of the specimens after removal and section.



FIG. 428.—Case 8. Papillary synovial inclusions without distension of cysts.

CASE REPORTS.

CASES REPORTED IN 1921.—

Case 1.—Male, age 40. Cyst of outer cartilage. Operated upon eighteen months previously because of pain and local swelling. Cyst alone removed. Swelling recurred after six months, and pain necessitated a second operation at which I removed the entire cartilage. Left knee.

Case 2.—Male, age 21. Cyst of outer cartilage. Removal of cartilage and cyst. Left knee.

Case 3.—Male, age 26. Cyst of outer cartilage. Removal of cartilage and cyst. Left knee.

CASES NOT PREVIOUSLY REPORTED.—

Case 4.—Male, age 22. Injury to right knee six months previously. Swelling the size of a cherry over outer cartilage. Cartilage and cyst removed. (Figs. 425, 429.)

Case 5.—Female, age 32. Hit by a truck on the outer side of the right knee a year before the appearance of a swelling. Pain for three months and a feeling of stiffness in the joint. Limitation of extension at 170°. Removal of cyst and cartilage.

Case 6.—Male, age 46. History of a fall and injury to right knee three years before the present swelling appeared. This has been noticed for five months. Pain on extension. Removal of outer cartilage and cyst.

Case 7.—Female, age 42. Three months' history of pain in the left knee, with the appearance of a swelling on the outer side soon after. The swelling is tensely fluctuant. There is evidence of early osteo-arthritis in both knees. Removal of cartilage and cyst. (Figs. 423, 430.)



FIG. 429.—Case 4.

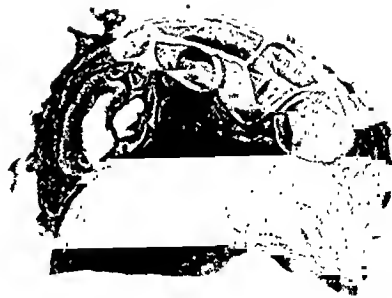


FIG. 430.—Case 7.

Case 8.—Male, age 30. Six months previously left knee injured at football. Tense cyst over external cartilage. Removal of cartilage and cyst. (Fig. 428.)

Case 9.—Male, age 22. Pain in joint, with local swelling over outer cartilage. Swelling is the size of a walnut. Injury at football six months previously. Removal of cartilage and cyst. This man is playing active football again and has had no further trouble with the knee.

Case 10.—Female, age 24. A blow on the left knee several months ago was followed by a swelling. Pain has been present, more or less continuously, for

three months. Swelling varies in size from time to time. Removal of outer cartilage and cyst.

Case 11.—Male, age 31. Right knee injured on active service. Pain and swelling over outer side. Removal of cartilage and cyst. (Figs. 422, 424.)

Case 12.—Male, age 10. Swelling over inner side of left knee for one year, said to have followed on a blow on the side of a desk at school. Painful at first, but less so now. Growing in size. Removal of cartilage and cyst. (This boy was shown to the British Orthopædic Association at Manchester in October, 1925, prior to operation.)

Case 13.—Female, age 28. Right knee twisted two years previously. Swelling on outer side of joint noticed about ten months ago. Tense fluctuant swelling. Removal of cartilage and cyst.

Case 14.—Male, age 6. No history of injury. Swelling over inner side of joint for several months; getting larger. Very little pain or tenderness on pressure. Removal of cyst only. Up to date (November, 1928) no sign of any recurrence. Operation was on Feb. 15, 1926. (Fig. 431.)



FIG. 431.—Case 14.

Case 15.—Male, age 20. Knee injured at work four months ago. Swelling over outer cartilage the size of a filbert. Removal of cyst and cartilage. (Figs. 427, 432.)

Case 16.—Male, age 23. Football injury with history of attacks of pain and swelling subsequently. Inner side of joint. Removal of internal cartilage and cyst.

Case 17.—Male, age 33. No history of injury. Swelling over inner side of left knee. Painful and cystic. Four months' history. Removal of cartilage (internal) and cyst.

Case 18.—Male, age 22. Right knee twisted at football six months ago. There is a cystic

swelling over the outer cartilage which is loose enough to produce the well-known 'snap'. Excision of outer cartilage and cyst.

Case 19.—Male, age 54. History of several injuries in coal-mine. Right knee. Five months ago noticed a swelling over the outer side: painful. Cystic swelling. Knee moves through full range. Slight osteo-arthritis present. Removal of cartilage and cyst.

Case 20.—Male, age 19. Six months' history of pain in left knee. No very definite injury. Knee 'gives way' when walking. Swelling the size of walnut over outer cartilage. Removal of cartilage and cysts. (Figs. 420, 421, 426.)

Case 21.—Male, age 24. Seven months previously hit the right knee on a wagon. Since then has had pain in the outer side. Swelling over outer cartilage about half an inch in diameter, tender on pressure. Removal of cartilage and cysts.

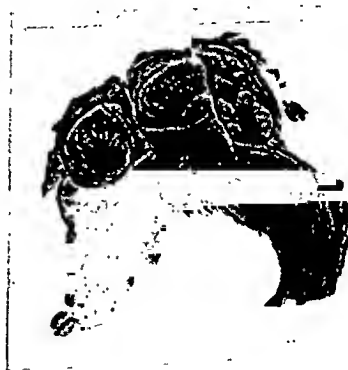


FIG. 432.—Case 15.

SUMMARY OF TWENTY-ONE CASES.

Males	17
Females	4
External cartilage affected	17
Internal cartilage affected	4
Left knee affected	12
Right knee affected	9

CONCLUSIONS.

1. I report 18 additional cases of semilunar cartilage cyst; 14 in the external and 4 in the internal cartilage.
 2. Cysts of the cartilages occur in either cartilage in the proportion of external 5·25 to internal 1.
 3. They are by no means infrequent.
 4. An associated history of injury occurs too frequently to be neglected as a cause.
 5. Endothelial cells can in most cases be demonstrated lining one or more of the cysts.
 6. The cysts are probably of developmental origin.
 7. Prognosis, after removal of cyst and cartilage, is excellent.
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REFERENCES.

- ¹ OLLERENSHAW, R., *Brit. Jour. Surg.*, 1921, viii, 409.
- ² EBNER, A., *Münch. med. Woch.*, 1906, xxix, 1737.
- ³ SCHMIDT, *Ibid.*, 1415.
- ⁴ RIEDEL, *Deut. Zeits. f. Chir.*, 1915, cxxxii, 167.
- ⁵ EDEN, *Naturwiss. med. Gesell. zu Jena*, 1911, May 18.
- ⁶ KLEINBERG, S., *Jour. Bone and Joint Surg.*, 1927, April.
- ⁷ ALLISON, N. and O'CONNOR, D. S., *Surg. Gynecol. and Obst.*, 1926, Feb., xlii.
- ⁸ BRISTOW, W. R., *Robert Jones Birthday Volume*, 1928. London: Humphrey Milford.
- ⁹ McMURRAY, T. P., *Ibid.*
- ¹⁰ ZADEK, I., and JAFFE, H. L., *Arch. of Surg.*, 1927. Nov., 677.

DEFORMITIES OF THE LUMBOSACRAL REGION OF THE SPINE.*

By JAMES F. BRAILSFORD,

ASSISTANT RADIOLOGIST AND CLINICAL LECTURER IN RADIOLOGY TO THE QUEEN'S HOSPITAL,
AND HONORARY RADIOLOGIST TO THE ROYAL CRIPPLES HOSPITALS, BIRMINGHAM.

SUMMARY OF CONTENTS.

- I.—ANATOMICAL CONSIDERATIONS.
- II.—RADIOGRAPHIC TECHNIQUE.
- III.—DEFORMITIES DUE TO DEVELOPMENTAL ABNORMALITIES
- IV.—SPONDYLOLISTHESIS.
- V.—DEFORMITIES DUE TO INJURY.
- VI.—DEFORMITIES DUE TO PATHOLOGICAL CHANGES IN THE BONES AND JOINTS.
- VII.—CONCLUSIONS.

THE radiographs of over three thousand patients attending several different types of hospitals have been studied during the past two years, and a clinical examination has been made of those patients whose radiographs showed any deformity of the lumbosacral area. In addition the lumbosacral area of forty post-mortem subjects has been investigated by dissection and X rays; and the specimens in the Museums of the Royal College of Surgeons and the University of Birmingham have been examined and photographed. To illustrate the cause of the deformities, radiographs of typical cases are used; photographs are employed to show characteristic features exhibited by deformed patients.

I. ANATOMICAL CONSIDERATIONS.

In the foetus only the thoracic and sacral curves are present in the spinal column, but as the child assumes the erect posture, two secondary curves develop—the cervical and the lumbar. Normally until puberty, when standing erect, these primary and secondary curves pass almost imperceptibly into one another, preserving the balance of the trunk by concavity alternating with convexity.

The forward lumbar curve, which is more pronounced in the female than the male, begins at the middle of the last thoracic vertebra and ends at the anterior surface of the lumbosacral joint. This point, which in the erect posture is said to be vertically under the occipital condyles, marks the commencement of the sacral concavity—a concavity which faces downwards and forwards, and extends to the tip of the coccyx. As adult stature is reached,

* Paper awarded the Robert Jones Gold Medal and Prize of the British Orthopaedic Association for 1927.

the constant pressure of the weight of the trunk increases the sharpness of the lumbosacral angle. This weight is borne by the opposing surfaces of the vertebral bodies and their intervertebral discs anteriorly, and to a lesser extent by the articular processes posteriorly. In the healthy adult with a normally developed spine the curvatures are such as allow these surfaces to take the bulk of the downward pressure, and the erect posture is maintained by the tone of the musculature, without strain being placed upon the muscles and ligaments of the vertebral column. If, owing to ill health, the tone of the musculature is diminished, the weight causes an exaggeration of the normal curvatures and throws a strain upon the ligaments and articular surfaces. This is of course most marked at the lowest part of the trunk, where the flexible upper part articulates with the rigid extremity—that is, the lumbosacral joint. Similarly, if the bony structures of this area have a congenital weakness or their strength is diminished by any pathological process, the superincumbent weight will produce deformities of a nature dependent upon the position and extent of the weakness or lesion. Before considering abnormalities it will be well to describe briefly the chief distinctive characters in the normal anatomy of the lumbosacral area.

The 5th lumbar vertebra has several features which distinguish it from other lumbar vertebrae. Its body is slightly wedge-shaped, being deeper anteriorly than posteriorly. Its transverse processes are continuous with the anterior surface of the vertebral body, and in relation to the body lie on a more anterior plane than the transverse processes of the vertebrae above: they are usually shorter than the 3rd lumbar transverse processes. Professor Brash points out that there is a gradual increase in the size of the transverse processes of the lumbar vertebrae from the 1st to the 3rd; that the 4th are shorter, and the 5th usually a little longer than those of the 4th, and point upwards, outwards, and backwards. Professor Fawcett points out that the transverse processes of the 5th lumbar vertebra have two epiphyses—one for the costal element and one for the transverse element.

The spinous process is usually a little shorter, more pointed, and not so deep as the spinous processes of the other lumbar vertebrae, and its extremity is on practically the same plane as the inferior border of the vertebral body. The pedicles are broader, being strengthened by the continuation of the transverse process into the body. The superior articular surfaces are wider apart than those of the upper vertebrae. The inferior processes are about as wide as the superior processes. There is a great variation in the direction in which the articular surfaces face; in most cases the superior facets face backwards and slightly inwards, the inferior facets backwards and slightly outwards. The intervertebral disc of the lumbosacral joint is also wedge-shaped and thicker anteriorly than posteriorly, thus making allowance for the lumbosacral angle.

The five sacral vertebrae have fused together into a triangular-shaped mass. The costal elements are represented by the row of small tubercles on either side of the posterior surface. The spinous processes are smaller than those of the lumbar vertebrae. The laminae of the lower vertebrae often fail to fuse and complete the neural arch. The sacrum has a concave anterior face which looks forwards and downwards. The superior articular facets usually face backwards and inwards, but these also show many variations.

The opposing surfaces of the 5th lumbar vertebra and the sacrum are joined together to form the lumbosacral joint by the following ligaments: (1) Intervertebral fibrocartilage, which also acts as a buffer between the vertebral bodies. (2) Anterior longitudinal ligament, which is attached to the intervertebral fibrocartilage and the edges of the vertebral bodies—the fibres, being of various lengths, joining two, three, or more vertebræ. (3) Posterior longitudinal ligament, which is attached to the posterior surfaces of the adjacent margins of the vertebral bodies. It is narrower opposite the bodies and therefore has a dentate appearance. (4) Articular ligaments and capsules. (5) Ligamenta flava—yellow elastic fibres which bind together the laminae of the adjoining vertebræ and close the intervals between the laminae; the fibres extend laterally as far as the articular capsules; medially they meet under cover of the root of the spinous processes. (6) Interspinous ligaments, which are obliquely interlacing fibres that pass from adjacent spinous processes from the tip of one to the base of the next. (7) Supraspinous ligaments—from the tip of one spinous process to the other. (8) Lateral lumbosacral ligament, which passes from the transverse process of the 5th lumbar to the base of the sacrum, expanding as it proceeds; some fibres are attached to the anterior sacro-iliac ligament. (9) Iliolumbar ligament from the tip of the transverse process of the 5th to the iliac crest. Some fibres from the 4th lumbar transverse process blend with the fibres from the 5th. The iliolumbar band proper is a thickening of the ventral layer of the lumbar fascia. (*Quain's Anatomy.*)

The psoas muscle passes over the antero-lateral aspect of the lumbosacral joint. The posterior relations are the intertransversarii, interspinales, multifidus, sacrospinalis, and the lumbodorsal fascia. The quadratus lumborum is attached to the transverse processes. In antero-lateral relation to the joint on each side is the lumbosacral trunk, which comprises the whole of the anterior division of the 5th and a part of that of the 4th lumbar nerve: it appears at the medial margin of the psoas major and descends over the pelvic brim and in front of the sacro-iliac joint to join the 1st sacral nerve. The last lumbar nerve emerges between the upper sacral articular process and the back of the intervertebral disc, and its posterior primary division turns back round the process to reach the postvertebral muscles, while its anterior part runs forwards, outwards, and downwards on the ala, being joined here by the branch from the 4th. Thus the nerve appears to emerge through an osseofibrous foramen completed externally by arching fibres of the iliolumbar ligament. All these structures are placed deep in the psoas. (*Frazer.*) Other structures in antero-lateral relation to the joint are the common iliac arteries and their bifurcations, the iliac veins, and the ureters. The pelvic colon and the mesocolon are also closely related to the left antero-lateral aspect.

The 5th lumbar nerve passes through an oblique bony canal which is directed forwards and outwards and is bounded in front by the postero-lateral surface of the body of the 5th lumbar vertebra, the posterior surface of the intervertebral disc, and the posterior surface of the upper part of the sacrum; laterally by the psoas muscle and some fibres of the iliolumbar ligament; and posteriorly by the anterior surface of the articular processes.

The importance of these joint relations to the nerve canal are emphasized in cases where the joints are involved in arthritic changes. Boniot and

Forestier state that the 4th and 5th bony nerve canals are long and that the nerve funiculus is not protected by an anatomical arachnoid sheath and is therefore influenced and irritated by variations in the rich venous plexus contained in the bony canal.

Causes of Lumbosacral Deformities. — The deformities of the lumbosacral region of the spine are due to: (1) Congenital abnormalities; (2) Injury; (3) Pathological processes involving the bones, ligaments, muscles, and nerves of the part; (4) Habitual faulty posture. The degree of deformity depends on the nature and extent of the lesion. It reveals itself to the examining surgeon as a faulty posture or gait; obliteration, exaggeration, or reverse of the normal antero-posterior spinal curvature; the production of a lateral curvature; the presence of a tumour; and the limitation of movements. The same type of deformity may have a widely different etiology, and even after a thorough clinical and X-ray investigation the true nature of the condition may not be determined. The clinical examination may indicate the nature of the lesion, but the bone and joint changes may not be of sufficient magnitude to show on a radiograph; sometimes a radiograph will show that the lesion is greater and more extensive than the clinical examination would lead one to suppose, and may throw much light on the etiology.

It is therefore the common practice of orthopædic surgeons to supplement their clinical examination by an X-ray investigation. For this reason it is considered advisable to give a few details of the necessary X-ray technique which the radiologist should observe.

II. RADIOGRAPHIC TECHNIQUE.

The radiographs should be of good quality, and should include both antero-posterior and lateral planes, while in some cases additional valuable help may be obtained from stereoscopic pictures taken with a transverse or longitudinal shift of the tube, and from radiographs taken in oblique planes, such as the plane of the lumbosacral joint—that is, with the central ray parallel with the inferior surface of the



FIG. 433.



FIG. 434.

5th lumbar vertebra, the tube pointing obliquely in the proximal direction with an angle of 40° from the vertical. *Fig. 433* is an ordinary antero-posterior radiograph of the lumbosacral area. It shows an apparent crowding together of the 5th lumbar and 1st sacral vertebrae owing to a sharp lumbosacral angle. *Fig. 434* is from the same patient taken with the central ray in the plane of the lumbosacral joint. It shows that these two vertebrae are normal

in shape and that the lumbosacral interspace is not diminished. Le Wald has pointed out that radiographs such as *Fig. 433* have been interpreted as showing the condition of spondylolisthesis.

There is a temptation in hospital practice to be satisfied with only an antero-posterior radiograph if this does not show any obvious defect: this practice probably originated in the 'pre-Potter-Bucky and rapid film days', when it was difficult and in some cases impossible to obtain lateral radiographs. Even as late as 1925 Turner and Tchirkin stated in an article, "owing to difficulties in radiography we are unable to present a lateral radiograph of the spine."

The pictures should be taken with a tube giving the softest X rays consistent with the necessary penetration, so as to obtain the maximum of contrast in the radiograph; and the exposures should be as rapid as possible to eliminate the blurring effect of movement and the danger to the skin of the patient.

With children and thin patients the best-detailed pictures are obtained by rapid exposures to a soft gas tube on duplitized films between intensifying screens without the Potter-Bucky diaphragm, which separates the patients from the film by an interval and just robs the radiograph of its sharpness and fine detail; but for the average adult and stout patients the Potter-Bucky diaphragm is essential. A narrow cylindrical tube-box diaphragm will screen off other secondary rays and help to give a clearer picture.

III. DEFORMITIES DUE TO DEVELOPMENTAL ABNORMALITIES.

The variations in the development of the elements of the lumbosacral region of the vertebral column are very numerous, and I shall deal with their importance in producing deformity according to the position of the variation. The following anomalies were found:—

			Per cent
Spina bifida occulta—5th lumbar	6
Spina bifida occulta—1st and 2nd sacral..	11
Sacralization of 5th lumbar—one side	3.4
Sacralization of 5th lumbar—two sides	4.7
Hemivertebrae	0.3

These figures may be compared with those of other workers. O'Reilly, who examined a large series of cases with anatomical variations, considers that 50 per cent of them showed symptoms of varying severity. Pugh, on the other hand, states that 85 per cent of the backs which he has examined showed some deformity, and he therefore concludes that the majority have no clinical significance. Rich examined 300 cases of painful back and found that 30 per cent showed some deformity or malposition of the 5th lumbar vertebra.

The Vertebral Body.—The bodies of the vertebrae develop from one centre of ossification, which appears at about the fifteenth week. An epiphysis for the superior and inferior surface appears about puberty and unites with the main body at about the twenty-eighth year. In some cases the centres appear which develop into hemivertebrae and do not fuse, thus giving the X-ray appearance of a split body. This is shown in *Fig. 435*, which illustrates a congenital 'split' of the 12th dorsal vertebra with some separation of the two halves. This appearance should not be mistaken for fracture.

In other cases only one of the two lateral centres develops. This may remain as a separate unit or it may fuse with the elements above or below; but whereas the 'split' body does not give rise to any deformity, as a rule the lateral hemivertebrae produce a lateral curvature. We have seen that the normal spine forms a series of antero-posterior curves, but with this asymmetrical growth the mechanics of the balance of the erect posture will be altered and a compensating lateral curvature must be formed. This cannot occur without a rotation of the other vertebrae, as was demonstrated by Lovett, who showed that a flexible straight rod could be readily bent in either an antero-posterior or a lateral direction—whereas a curved rod could only be bent in another plane if the rod underwent some rotation.



Fig. 435.

Fig. 436 shows the scoliotic deformity produced by a hemivertebra of the 1st sacral segment and an irregular development of the left pedicle and inferior articular processes of the 3rd lumbar vertebra and the superior articular processes of the 4th lumbar vertebra, which give the appearance of a fracture of the 4th vertebral body. Professor Dwight also mentions a case of an extra half segment in the sacrum. Professor Brash suggests that in the case of hemivertebrae the body condition is secondary to the nerve abnormality.



Fig. 436.

There is a specimen (No. 322) in the Museum of the Royal College of Surgeons, of the lumbar spine and sacrum of a child, age 12, in which the 5th lumbar is represented by a right-sided hemivertebra, and the axes of the 1st and 2nd sacral vertebrae are

poorly developed. This has caused a very marked angular curvature of the sacrum to the left, which is at right angles to the lumbar vertebrae. The laminae of the vertebrae from the 3rd lumbar to the extremity of the sacrum have not fused and their extremities are wide apart, indicating an extensive spina bifida as well. This specimen is described by Parker in the *Transactions of the Clinical Society*, 1885.

In some cases two vertebral bodies fuse together, but as a rule these are only discovered by accident when the spine is X-rayed for some other condition. This happened in the case of the patient whose radiographs are shown in Figs. 437 and 438.



Fig. 437.



Fig. 438.

What appears on a radiograph as an almost shapeless mass may develop instead of the normal lumbar vertebræ. *Figs. 439-442* illustrate the abnormal ossification of the lumbar spine of a male child, age nearly 5 years. The normal spine appears to cease at the 1st lumbar, after which three or four shapeless bodies are seen, with no attempt at neural-arch formation. The sacrum is represented by a small shapeless structure between the iliac bones. The child shows a marked kyphosis of the lumbar area and deformities of the lower extremities, including talipes and webbing of the knees and toes; there is also incontinence. There is no external appearance of *spina bifida*. Bowman quotes a case of a boy, age 6, without a sacrum or coccyx.

The Articular Processes. — The articular facets are developed from the ossification centres of the neural arch—one on either side of the body. Rambaud and Renault are of the opinion that the arch has two primary centres on each side and the two parts in each half are united by a plate of cartilage set obliquely between the superior and inferior articular processes. This may explain those examples of 5th lumbar vertebræ

which have a separate neural arch comprising the spinous process, laminae, and the inferior articular processes which are united during life by synchondroses. No suggestion of this type of deformity may be obtained from the radiograph.

There is a specimen in the Royal College of Surgeons Museum which shows a well-developed neural arch, including thick laminae and normal-sized inferior articular processes and spine of the 4th lumbar vertebra, as a separate unit, and not fused with the remainder of the vertebra.

Figs. 443 and 532 show such a deformity of the 5th lumbar vertebra, and



FIG. 439.



FIG. 440.



FIG. 441.



FIG. 442.

Figs. 444 and 445 are radiographs of the vertebra shown in *Fig. 443*, with the units attached by soft wax, and demonstrate that even with the dried specimen the radiographs give no evidence that the laminae have failed to fuse. They also show that the two parts are fully developed.

The articular facets of the lumbosacral joints show great variation in their shape and in the plane of the articular surfaces. The superior articular facets on the majority of sacrum are slightly crescentic and face backwards and slightly inwards. If the greater part of the facet faces backwards, no idea of the condition of the surface of the joint can be obtained from a radiograph; but if the greater part faces inwards, then the joint surfaces with a narrow space between them can be seen on the radiograph and any irregularity of the surface may be detected. In some cases one facet faces backwards and the other inwards—what Putti describes as ‘anomaly of articular tropism’. As a general rule the surfaces are vertical, but this is not always the case. A



FIG. 443.—Fifth lumbar vertebra in which the arch and the body have not fused.



FIG. 444.—Antero-posterior radiograph of same specimen, with the arch and body united with wax. (Note that the non-union does not show.)

careful examination of all the radiographs of more than three thousand patients suggests that 57 per cent face backwards, 12 per cent inwards, and 31 per cent are mixed—that is, one side may face backwards and the other inwards; one may be vertical, the other oblique.



FIG. 445.—Lateral radiograph of same specimen, which shows no evidence of non-union of the neural arch and body.

In some cases the surfaces are quite flat and face directly backwards (see *Figs. 451 and 453*); this is said to be the rule in ‘native races’. Most authorities are agreed that with an asymmetrical development of the articular processes, abnormal movements occur. Obviously the flat type only gives stability in one plane, so that any force directed in another plane will strain the ligaments of the joint. Separation of the neural arch with the inferior

articular processes may lead to a forward dislocation of the lumbar spine on the sacrum—spondylolisthesis.

Willis found a separate neural arch in 31 out of 748 cases which he examined: in 23 it was bilateral; the right half was involved in every case; 30 were males, 1 was a female; 28 were white, 3 were negroes. Von Lackum studied 30 bodies to determine if any anatomical instability was present in the lumbosacral area to account for the prevalence of low back pain. He found that in only 6 were the articulations of the lumbosacral joint symmetrical: 18 were grossly asymmetrical.

In only three of the cases in my series was a clinical deformity explained

solely by an abnormal development of the articular surfaces. In these case the superior articular facets were small and irregular, while the inferior facets and laminae of the vertebra above showed abnormal development, which resulted in scoliosis (Figs. 446 and 447).

Fig. 446 is the spine

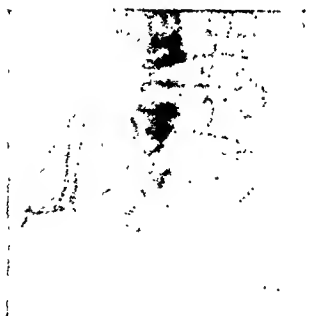


FIG. 446.



FIG. 447.

of a woman, age 35, who complains of pain round the lower right ribs and lower spine, which is of a constant aching character, worse when lying down or sitting than when standing. The radiograph shows a congenital deformity of the right side of the 4th and 5th lumbar vertebrae and 1st sacral articular processes, which has produced a scoliosis.



FIGS. 448, 449.—Showing asymmetrical facets of the lumbosacral joint.



FIG. 450.—Showing symmetrical facets of the lumbosacral joint.

Asymmetry of the articular facets may be due to any lesion producing scoliosis (see Figs. 446-450). In the case shown in Fig. 448 this asymmetry is associated with sclerosis of the bone surrounding the facets and the base of

the pedicle. It was due to atrophy of the left lower extremity following anterior poliomyelitis. *Figs. 451-454*, from photographs of actual specimens, may also be studied in this connection.



FIG. 451.—Sacrum with flat articular facets.



FIG. 452.—Sacrum with asymmetrical crescentic facets.

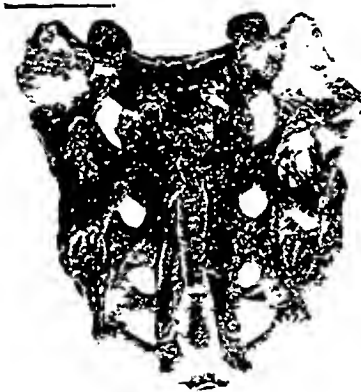


FIG. 453.—Posterior view of same specimen as *Fig. 451*. Note the flat articular processes and that the laminae of the sacrum have not met, and the canal is left open.



FIG. 454.—Posterior view of same specimen as *Fig. 452*. Note asymmetry of articular facets.

Defects in the Neural Arch.—The centres of ossification for the neural arch are present at birth and fuse together during the first year, and about the sixth year they fuse with the body of the vertebra. In the radiographs of the 3000 patients examined, defects in the neural arch of the 5th lumbar vertebra were shown in 6 per cent, and in the 1st and 2nd sacral in 11 per cent. The degree of defect varied. The patients presenting an obvious tumour associated with spina bifida were not radiographed as a rule. In some the neural arches of the lumbosacral area were represented by small tubercles, but the majority only showed a split between the posterior extremities of both sides, one or other side often having a bulbous extremity—representing that no bone union had occurred. This non-fusion of the laminae often gives an appearance suggestive of fracture—indeed, several of the cases had previously been reported as fracture of the laminae.

Sutherland found a failure of fusion of the 5th lumbar or 1st sacral in 5 per cent (1st or 2nd sacral 70 per cent, 5th lumbar 24.5 per cent). Baetjer found a failure of fusion of the 5th lumbar in 15 per cent, Wentworth in

8.1 per cent, and Willis in 4.28 per cent. The last-named described seven types of defect; some of these occurred singly, others showed several of the defects (*Fig. 455*).

Decker describes a 6th lumbar vertebra with a spine which is split into two unequal portions and an articular surface between. The left portion is

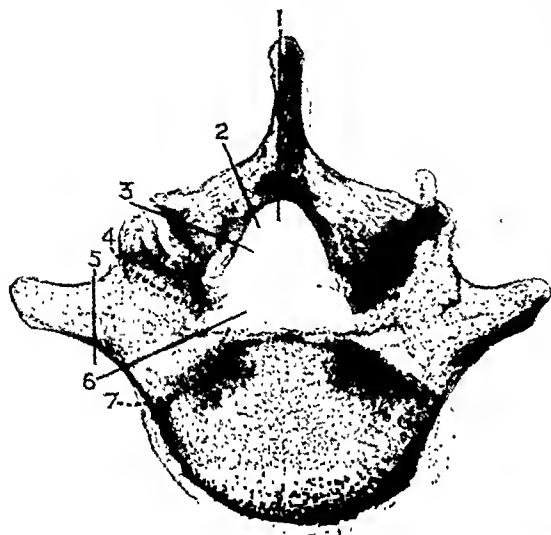


FIG. 455.—Showing the seven lines of non-fusion found by Willis in the examination of 748 spines.

shorter (1.2 cm. long), more or less pointed, and is attached to the lamina (1.5 cm. broad), and with the lamina is directed backwards and outwards so as to be slightly concave laterally. The right portion embraces most of the spine, is club-shaped, and is attached to the lamina (1.6 cm. broad), which is directed almost horizontally outward. Another defect in the neural arch occurs in the right pedicle just behind and below the superior articular process, where there is a narrow triangular articulation, 2.3 cm. by 0.6 cm. long, looking upwards and slightly forwards. The separation of the pedicles is not complete, however, for below this

joint, between it and the inferior articular process, there is definite bony continuity.

Spina Bifida.—Five different types of spina bifida are met with:—

1. *The Myelocoele*, in which the central canal of the cord is exposed, and the bone elements of the neural arch are represented by small nodules on either side of the posterior surface of the body. This type is usually located in the dorsi-lumbar area and is not compatible with life. It may extend along the whole spine.

2. *The Syringomyelocoele*, in which there is a dilatation of the central canal with stunted development of the laminae. In this type there is a tumour of variable size, and cord symptoms are present—paralysis of the lower extremities and incontinence.

3. *The Meningomyelocoele*, in which there is a dilatation of the meninges of the cord through which the fibres of the corda equina pass. This is a very common type and is usually accompanied by paralysis of the lower extremities and incontinence, sometimes by rigidity and contractions.

4. *The Meningocele*, which is a dilatation of the meninges, and contains only the cerebrospinal fluid and no nerve elements; the common site is the lumbosacral region of the spine. Usually four or five of the neural arches are stunted. The cystic swelling may be as large as the child's head or quite small. It may be tense or flaccid, and the hole communicating between the cystic dilatation and the meninges may be very small. This and the preceding types are often associated with hydrocephalus. Operative measures applied to the

cystic swelling may be followed by the production of hydrocephalus. The symptoms may be slight, such as weakness of the lower extremities and sphincters, but may on occasion be severe. Whitehead records a spina bifida in an adult female which, from birth to the age of 21, was associated with a meningocele about the size of an egg, but gave rise to no symptoms. At this time the patient's circumstances compelled her to do hard manual labour and the tumour increased to the dimensions of 22 inches in circumference, and concomitant with the increase in size she commenced to have severe headaches, nausea and vomiting, attacks of vertigo, and transient moments of defective vision whenever the tumour was subjected to pressure, even that of clothing. The tumour was aspirated, cauterized, and after severe inflammatory changes finally healed, and the patient made a complete recovery.

5. *Spina Bifida Occulta*, which is the commonest type. In this condition there is a failure in the development of the neural arch of several vertebræ—usually in the lumbosacral area. The only physical sign may be a small lipoma, or a tuft of hair, or a dimple in the skin. There may be no symptoms, but some cases show progressive deformities of the lower limbs, talipes equinus, pes cavus, or instability of the lumbosacral region with low back pain, or pain from pressure on the nerve trunks. Operative measures for removal of the cystic swelling or other abnormality may not influence the chief symptoms due to nerve abnormality, which may be in the same developmental segment and consequently at a much higher level in the adult cord. These developmental errors are usually symmetrical, but some cases show definite asymmetry. Mr. Fitzmaurice Kelly recently showed a case of a female child, age 4 years 4 months, in which the lateral mass of the sacrum and the 5th lumbar were missing, and a cystic mass was situated under the gluteus maximus. The child had severe paralytic talipes of both feet and incontinence of urine.

Particulars of a specimen of spina bifida associated with hemivertebra are given on page 567. Figs. 456-459 show different types of non-fusion of the



FIG. 456.
Symmetrical laminae.



FIG. 457.
Asymmetrical laminae.



FIG. 458.
Asymmetrical laminae.

laminae. Figs. 460 and 461, which are taken from a girl, age 7, show an extensive spina bifida from the 3rd lumbar to the extremity of the sacrum.

The lateral radiograph shows large articular processes of the 3rd lumbar. The patient exhibited no external evidence of spina bifida. A 'knuckle' kyphos was present, which was thought to be due to caries of the spine.



FIG. 459.—Symmetrical laminae with separate spinous process of 1st sacral.



FIGS. 460, 461.—Spina bifida extending from 3rd lumbar to the extremity of the sacrum.

Fig. 462 is the photograph of an external tumour in a man of 57. Radiographs showed only a faulty development of the 3rd sacral vertebra.



FIG. 462.

Spinous Processes.—The spinous processes are formed by fusion of the two sides of the laminae, which takes place in the first year, and their development is completed by the growth of an epiphysis which appears about the sixteenth year and fuses with the main mass in the twenty-fifth year. In some cases, where the laminae fail to fuse, the epiphysis for the spinous process remains as a separate bone and can be seen on many radiographs of the lumbosacral area (see Fig. 459).

The spinous process of the 5th lumbar shows variation in shape, size, and position. Sometimes it is flattened, drawn out, and, instead of being shorter than the spinous process of the 4th, it may be longer, as in the cases of spondylolisthesis. It may be

united only to one side of the neural arch, or may be represented by the posterior extremities of the laminae which have come together but not fused (see Figs. 456–459). It may be directly in the mid-line or be directed to one side. If for any reason the lordosis of the lumbar spine is increased, or the lumbosacral angle is diminished, the spinous processes are approximated to one another, until in some cases they touch each other, and their opposing surfaces may be moulded or faceted. Obviously, with such a spine, extension is checked by the locked-home position of the spinous processes, which are subjected to an abnormal strain, and bursa and arthritic changes readily

develop (*see Fig. 539*). Such patients have pain in the back when standing erect, which is relieved by bending forward.

Transverse Processes of the 5th Lumbar Vertebra.—There is no more variable part of the 5th lumbar vertebra than its transverse processes and no part in which developmental asymmetry produces deformity in so many cases. Bound up with the irregular development of these processes is the numerical variation of the vertebral column and sacralization of the 5th lumbar or lumbarization of the 1st sacral. Numerous observers have studied this question. The normal formula—cervical 7, dorsal 12, lumbar 5, sacral 5, and coccyx 4—varies in about 20 per cent of cases (Quain).

The cervical region is the most constant, while the most frequent site for the variation to be noticed is the lumbosacral. This is due to the marked difference in the characters of the free lumbar vertebræ and the fixed consolidated sacral vertebræ. Attention is drawn to the variation in the cervical area by the cervical 'rib' pressing upon the brachial plexus or some of its elements, or the vessels. In the lumbosacral area variation is common; in my series 3·4 per cent show sacral characters on one side and lumbar characters on the other. The variation in this area appears to be more frequently in the direction of elongation rather than shortening of the column, but the statistics of different observers show differences in this respect. It will be noticed as a rule that when the change of character is at the junction of one series of vertebræ with another, the next junction will show a change in the same direction—a sort of oscillation passing distally or proximally.

In the case recorded by Professor Brash with the formula C6½, T13, L5, S5, C4, there was no evidence of the 5th lumbar vertebra having been derived from the sacrum. Failure of the 1st sacral segment to fuse with the other bodies may suggest a 6th lumbar (*see Fig. 433*). Six lumbar vertebræ is a common X-ray finding. Bardeen, after a study of reports of 1059 specimens including 75 of his own, found that numerical variations occur in about 16 per cent of vertebral columns, of which 7·5 per cent have compensated variations and 8·7 per cent uncompensated, equally divided between increase and decrease of segments. He says that there is no evidence that intercalation of a whole vertebra occurs.

Side by side with these oscillations in the vertebral characters, variations are seen in the constitution of the lumbosacral plexus. Eisler records concomitant variations in the plexus of 18 per cent of the cases. They occur within wide limits, as shown by the following table of extreme cases.

	PRE-FIXED VARIETY	NORMAL	POST-FIXED VARIETY
Nervus furcalis ..	L3 & 4 (double)	L4	L5
Obturator	L1, 2, 3 ..	L2, 3, 4 ..	L2, 3, 4, 5
Femoral	T12, L1, 2, 3, 4	L2, 3, 4 ..	L2, 3, 4, 5
Tibial	L3, 4, 5, S1, 2	L4, 5, S1, 2, 3	L5, S1, 2, 3, 4
Common peroneal..	L3, 4, 5, S1	L4, 5, S1, 2	L5, S1, 2, 3

The transverse process is developed from the centre of ossification for the neural arch, but at the sixteenth year an epiphysis appears (sometimes

two, Fawcett) which fuses with the main process about the age of twenty-five. We have seen that the costal rudiments of the 5th lumbar extend over the pedicles to the sides of the body, whereas in the sacrum they enlarge and form the alæ on each side—the transverse processes only being represented by nodules on the posterior surface. A foramen has been observed in the 5th lumbar transverse process by Szawłowski and Dwight which marks the costal from the true transverse element—the anterior part of the transverse process being the homologue of a rib; the true transverse element is represented by the posterior part of its root, including the accessory process. When the transverse processes are large they may articulate with the sacrum—the lumbosacral transverse articulation of Dwight—but in some cases the processes grow out in a direct lateral line and articulate with the ilium.

There are marked differences to be seen in the breadth of the sacrum and the height at which it articulates with the ilium, apart from sexual differences. *Fig. 463* shows the sacrum with its superior border almost on the same plane as the iliac crests: a line joining the latter would pass through the 5th lumbar vertebra. The straight and long transverse processes of the 5th lumbar should be noted. *Fig. 464*, on the other hand, shows a low sacrum with the



FIG. 463.



FIG. 464.

ilium on both sides rising up to the height of the 3rd lumbar vertebra. The transverse processes of the 5th lumbar are short and turned upwards—the sacrum is narrow. The radiographs were taken in the same relative positions. The high type of sacrum is regarded by some authorities as unsound architecturally as

it is not so well supported by the sacro-iliac and spinal muscles and ligaments, but radiographically it appears more sound; the lumbosacral angle is a gentle curve extending over several vertebræ. The same condition is seen with complete symmetrical sacralization of the 5th lumbar.

Moore is of the opinion that anatomically and architecturally the sacralized formation appears stronger and more able to resist strain than the usual arrangement. Von Lackum suggests that surgical measures to fuse the 5th lumbar with the sacrum would be better than removing interfering masses. While this is so with symmetrical sacralization, with asymmetrical development of the 5th lumbar processes curvature and rotation of the lumbar vertebræ is produced which in time is followed by compensating curvature of the dorsal vertebræ in the attempt to preserve the balance of the erect posture. The vertebræ in their normal positions are so constructed that they can withstand the strain of normal movements and postures, but in the positions which they occupy as the result of the curvature they are placed at a disadvantage and cannot resist the strain put upon them. The curvature therefore tends to increase.

Figs. 465 and 466 are photographs of the anterior and posterior surfaces of a specimen of unilateral 'lumbarization'. It will be seen that the lower four segments have fused to form the body of the sacrum. The upper segment shows sacral characters on the left side and lumbar characters on the right. The joint space between this segment and the main sacral body is



FIG. 465.—Anterior view.



FIG. 466.—Posterior view.

almost obliterated, the angle between them is 154° . The right transverse process resembles that of a normal 5th lumbar vertebra and is directed backwards and outwards; the left transverse process is higher than the right and extends further back, but its pedicle is greatly thickened on a more anterior plane ($\frac{1}{4}$ in.) and is continued forward by its costal element to articulate with the ala of the sacrum. The intervertebral canal on the left side is therefore longer and narrower; it is narrowed even more by the 'lipping' which has occurred at the transverse 'lumbosacral' joint. The body is wedge-shaped

in two directions, being deeper on the anterior and the left sides, and is twisted to the left. Its upper border is oblique and the articular facet is on a higher plane on the left. This marks the beginning of a scoliosis, as illustrated in *Figs. 467 and 468*. It will be noticed in *Fig. 466* that the neural arch of the upper segment has not fused, the right side being longer than the



FIGS. 467, 468.—Illustrate the beginning of a scoliosis produced by unilateral 'sacralization' of the 5th lumbar vertebra.

left and forming the spinous process, which is deviated to the left. The right superior facet of the fused body of the sacrum is normally developed, but the left is smaller and is continued proximally into an irregular process which is

partly fused with the poorly developed upper segment inferior articulation. The inferior articular processes of the upper segment are not so well developed or so far apart as the superior.

Figs. 469 and 470 show a bilateral 'sacralization' of the 5th lumbar vertebra in which the transverse processes have not developed symmetrically. The left is completely fused with the sacrum, the right has a definite joint surface



FIG. 469.



FIG. 470.

with the sacrum. There is a fusion of the left side of the bodies by new bone which protrudes into the intervertebral foramen on that side. The body is only slightly twisted to the left. The neural arch, including the spinous process, inferior articular processes, and lamina, is poorly developed and not fused with the superior articular processes or pedicles. The latter are continued distally as an irregular process to form an irregular joint with similar processes from the superior extremities of the superior articular facets of the sacrum, which are smaller and not so far apart as the superior facets of the 5th lumbar. The lumbosacral angle is 148° . The sacrum in this specimen is shown to consist of five bodies (cf. *Figs. 471 and 472*).

Figs. 471 and 472, which are photographs of a specimen from an Anglo-Saxon burial ground, show an asymmetrical 'lumbarization' of the upper sacral segment, only four segments being fused to form the main body of the sacrum. It shows similar features to the specimens already described (see *Figs. 465, 466, 469, 470*), but in this case the neural arch is fully developed. The spinous process is a little to the left of the middle line, the right lamina being a little longer than the left. The rotation of the body is less than in the preceding case. The intervertebral foramen on the right side, however, is not so deep as the left, which is again narrowed by the boss of new bone at the lumbosacral transverse joint. The inferior articular processes of the

upper segment are not so well developed or so far apart as the superior. The lumbosacral angle is 140° .

It will be noted in all the specimens that asymmetrical sacralization leads to a scoliosis. These specimens have completely fused on the one side, but the presence of an articular surface on the other side, together with the new bone formation at the site of the fusion, suggests that in earlier life movement did occur at this joint which was somewhat restricted and strained by the asymmetry of development and a tendency to one-sided fusion.

These specimens lend some support to the paragraph in *Orthopædic Surgery* by Jones and Lovett, and in Cunningham's *Anatomy*, which states, "the presence of a lumbosacral transverse articulation combined with osteoarthritis of the lumbosacral joint on the same side, may result in a reduction of the size of the intervertebral foramen which transmits the anterior division of the 5th lumbar nerve. Pressure on the nerve as it issues from this fibro-osseous foramen may account for the referred pain which is sometimes associated with these cases, whilst the branch of the 4th lumbar nerve which descends in front of the abnormal lumbosacral transverse articulation is also liable to be pressed upon." Usadel says that this suggestion is false. Putti supports the pressure-pain theory, but further states that "it must be borne in mind that the 4th lumbar contributes to the formation of the plexus, and it is often possible to demonstrate that the pain is due to involvement of this root and not the fifth, which alone could be affected by sacralization."

Baumen operated on twenty cases, in which he claims relief in practically all. He says: "In removing the deepest anterior part of a sacralized process there were distinct muscle twitchings, indicating the close proximity of a nerve cord (this has been observed several times), and as the last of the process was withdrawn, the nerve cord seemed to follow it up across the wound, until it occupied a position about one inch nearer the surface than it could have occupied before. This nerve cord must have been stretched tightly across the process and pressed upon by the process. The sciatic pain was relieved in this as in almost every one of the operated cases."

Aimes and Jaques, in 63 cases of persistent lumbar pain, noted 38 instances of sacralization of the 5th lumbar vertebra. Basset describes a case of a girl of 23 whose pain was permanently cured by a partial removal of the long transverse process of the 5th lumbar. Figs. 473 to 484 show different types of 5th lumbar transverse processes.

In my own series of cases 8.1 per cent showed sacralization of the 5th lumbar vertebra, 3.4 per cent being unilateral, 4.7 per cent bilateral. Many of these were discovered on the radiographs of patients complaining of symptoms suggestive of stone in the urinary tract for which they were X-rayed. There was no clinical evidence that the sacralization produced symptoms. In



FIG. 471.



FIG. 472.

a few cases, however, the patients complained of pain in the lumbosacral area, sometimes radiating down the back of the thigh, in which the only abnormality found was a sacralization. In one of these cases the patient had 'sciatica' with some atrophy of the gluteal muscles on the same side as the sacralization, which had produced a scoliosis. There seemed to be some



FIG. 473.—
Dichotomous branching of the left 5th lumbar transverse process, the costal element of which is articulating with the ala of the sacrum.



FIG. 474.—
Double fused transverse processes of 5th lumbar on both sides, with abnormal development of the other processes; also non-fusion of laminae of 1st sacral.



FIG. 475.—
A large left transverse process of 5th lumbar which is articulating with the ilium and sacrum. A lumbar curvature is associated with this abnormal development.



FIG. 476.—
Asymmetrical sacralization of the 5th lumbar on both sides. The right side has partly fused. Note definite scoliosis.



FIG. 477.—
Bilateral 'lumbarization' of 1st sacral vertebra with non-fusion of laminae. (Cf. with Fig. 478).



FIG. 478.—
Bilateral sacralization of 5th lumbar. There appears to be an over-development of the costal element with suppression of transverse element.

relation between the production of pain and the age at which articulation with the process occurred.

Figs. 479-484 are a series of radiographs of different patients to show the stages in the development of the unilateral sacralization of the 5th lumbar processes.

In children the large costal element of the 5th lumbar vertebra which is taking on sacral characters is often shown on the radiograph as a separate



FIG. 479.—
Sacralization
of right trans-
verse process of
5th lumbar.



FIG. 480.—
Sacralization
with marked
scoliosis.

bone, which suggests that it has a separate centre of ossification. Gegenbaur has pointed out that the anterior part of the lateral mass of the 1st, 2nd, and 3rd sacral vertebrae is developed from a separate centre which represents the costal element (Faweett). With unilateral sacralization there may or may not be scoliosis. If the lumbosacral transverse articulation is painful, the patient will list over to the opposite side and produce scoliosis. In those cases in which the 5th lumbar transverse processes are long and articulate with the ilium (Fig. 485), there may be no pain, but several cases which I have seen have complained of great pain over the articulation, and this has been the only bone abnormality to be seen on the radiograph to account for it.

Verrall states that in his experience when symptoms do occur they consist of intractable pain, both local and referred to the area supplied by the 4th lumbar nerve, as the junction between the 4th and 5th lumbar nerves



FIG. 481.



FIG. 482.



FIG. 483.



FIG. 484.

Figs. 481-484. —Radiographs showing different stages of unilateral sacralization of the 5th lumbar transverse processes.

lies immediately anterior to the process. He approaches the process by cutting a window from the ilium and thus removes the opposing surface.



FIG. 485.—Articulation of the 5th lumbar transverse processes with the ilium.

frequently show osteo-arthritic changes when other joints appear to be normal. Ossification of the iliolumbar ligaments may occur (Fig. 488), and this is usually associated with pain. Of the five cases in my series, all complain [of low back pain. In one case this was associated with pain in the

With erosion of the sacro-iliac joint, the surfaces may slip and the normal transverse processes of the 5th lumbar vertebra on one side may be brought into contact with the ilium and be a cause of pain when the original lesion is consolidated. The transverse process may also be brought into relation with the ilium by a scoliosis due to infantile paralysis, hip-joint disease, or any other lesion producing a curvature (Figs. 486, 487).

There is no doubt that with all these irregular and asymmetrical joints, unusual strain is put upon the opposing surfaces; adventitious bursæ form and from time to time become inflamed and give rise to pain. Such surfaces



FIG. 486.—Articulation of right transverse process of 5th lumbar with ilium due to sacro-iliac disease and scoliosis.



FIG. 487.—Articulation of left transverse process with ilium due to old tubercle of left hip-joint.



FIG. 488.

gall-bladder area, which led to some difficulty in the diagnosis. An X-ray examination revealed calcification of the iliolumbar ligaments and a collection of gall-stones. Doubt refers to twenty cases observed at the Henry Ford Hospital. He states that the calcified ligaments and transverse processes were removed in one case with complete recovery.

Lumbosacral Angle.—Bardeen points out that in the embryo of 5 cm. (9 weeks) the sacrum is nearly straight and in a line with the lumbar region. From this time onward the lumbosacral angle begins to form. When a child begins to walk the lumbar curve is formed

and the sacral concavity becomes more marked and the lumbosacral angle more salient. In the adult the lumbosacral angle is on the average 120° , but this shows variations in both directions; in the labouring class chiefly in the reduction of the angle. In the series which I have examined, those spinal columns with a high sacrum or with symmetrical sacralization of the 5th lumbar showed the widest angle. In these cases it can hardly be called an angle at all; the lumbar vertebræ and inter-vertebral discs are so shaped that the forward curve is a gradual one and the change to the sacral concavity is spread over several vertebræ. The anterior surfaces of the last lumbar and upper sacral segments if produced make an angle which may be as great as 160° . Commonly in the female the 5th lumbar body appears to be a little more wedge-shaped, and its spinous processes appear a little smaller to accommodate the greater sacral convexity.

The body of the 5th lumbar and the lumbosacral intervertebral disc are thicker anteriorly than posteriorly, as a result of which the weight of the trunk exerts a shearing strain at this joint and tends to diminish the angle. The actions of stooping, bending, sitting, and standing all call for lumbar movement; in fact, most of the principal movements of the trunk occur in the lumbar spine, flexion being the most extensive and more free in that region.

Considerable discussion has been taking place on the subject of different muscle functions. It is held by some that there are two different muscle functions—that of postural tone and that of movement. The former, controlled by the autonomic system, should maintain the correct position without effort or fatigue. When it fails in its function owing to some general weakness the other function of voluntary muscular contraction may be called in to take its place. This quickly leads to fatigue and pain. With this fatigue there is an exaggeration of the normal curves. The lumbar lordosis is increased and the intervertebral joints are maintained at the limit of their motion. This strain is usually felt by all patients after a prolonged rest in bed. A similar sequence of events is produced by regular employment which necessitates lifting or carrying heavy weights or assuming a bent position. As a result of this long strain in the fully flexed or extended position, the bones undergo a degree of moulding and the ligaments are stretched until eventually the muscles cannot correct the deformity. The balance, which in the normal subject is so well adjusted, is upset and a gradual increase in the deformity results. Goldthwait distinguishes two well-known variations from the normal skeleton which are particularly liable to suffer from lumbosacral backache:—

1. The tall, slender, visceroptotic type with a long narrow flexible spine, who often assumes a faulty posture. In this type the articular facets are flattened and movement is free. The lumbar lordosis is exaggerated and the lumbosacral angle is diminished. This results in approximation of the spinous processes to one another and adventitious bursæ are found between them. Constant slight traumata inflame the bursæ and they become a source of pain.

2. The short, thick, heavy type in whom the diarthrodial facets are large and crescentic. These limit the movements of the vertebræ, particularly lateral flexion. If the facets are unequal, the movements are irregular. In this type the lumbosacral angle is strained.

Sudden unexpected blows directed to the trunk usually cause damage to

the ligaments, as the muscles are not on their guard and the joints are forced to the extreme limit of their excursion; the ligaments controlling the extent of the movement are strained or even ruptured. As the lumbosacral joint is the junction of the mobile and the fixed parts of the vertebral column, any such downward thrust on the upper part of the trunk, or the momentum of a fall in which the patient lands on the buttocks, will strain or rupture the ligaments of the joint. If the part is placed at rest until the ligaments are healed, little harm may result, but if there is a repetition of small degrees of trauma of this nature, the ligaments will be permanently altered, the lumbosacral angle will diminish, and arthritic changes will occur in the joints.

Herndon examined 498 cases of injuries to the back, 68 per cent of which he diagnosed as sprain, the most frequent site being in the lumbosacral region.



FIG. 489.



FIG. 490.



FIG. 491.

The symptoms in these cases were pain, tenderness, stiffness, and limitation of movement. An antero-posterior radiograph of this condition may show

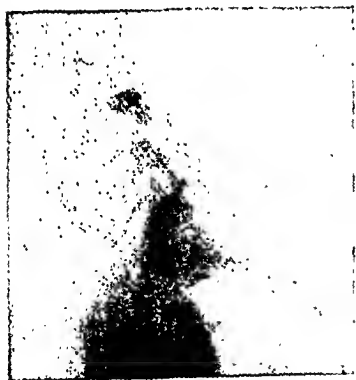


FIG. 492.

a tilting upwards of the spinous processes and an apparent crowding together of the vertebræ. This may be interpreted as a crushing or slipping forward of the vertebral bodies. A lateral radiograph will show that the appearance is due to a narrowing of the lumbosacral angle. A. Whitman has described five cases of this nature. They complained of pain in the back in the region of the lumbosacral joint; occasionally the pain radiated down the front and back of the thigh. The character of the pain varies from a sharp agonizing variety to chronic weakness and discomfort. He calls the condition a 'pre-spondylolisthesis'. Hibbs's fusion operation was performed in one case to prevent

an increase of the deformity. Figs. 489-492 illustrate normal variations of the lumbosacral angle. Figs. 515 and 516 show the condition of pre-spondylolisthesis (see p. 598).

IV. SPONDYLOLISTHESIS.

This is a term derived from the Greek word meaning 'gliding of a vertebra', and was first applied to the condition about to be described by Killian in 1852. The malady had been observed and described previously by Herbineau (1782), Rokitanski (1839), and Belloc (1849), but it was not until Neugebauer in 1892 published his description of 101 cases, for the most part museum specimens, that much attention was paid to the condition. Its chief importance, even at this time, was due to the difficulties which the deformity produced in obstetrics. Many other observers—notably Lane, Bowman, Darling, Lovett, Kleinberg, Balensweig, Turner and Tehirkin, Napier, Albee, Wideroe, Wiemens, Magnuson, Strasser, Barnes, Herrgott, Braun, Swartz, Spaeth, Roberts, Fitch, Palmer, and Lamb—published a series of cases.

All the earlier workers looked upon the condition as being peculiar to the female sex, but Codivilla (1908), Brandenburg (1910), and McCordiek (1912) reported a similar deformity in males. Many of the later writers are now trying to make out that the condition is a very common one, and is much more common in males than females. Thus Bowman says it is a common cause of low back pain, while Sir Arbuthnot Lane goes so far as to say that spondylolisthesis is the normal condition in coal heavers. Further, each writer appears to crowd into his list of cases a number in which the evidence of spondylolisthesis is very doubtful. Thus Albee, referring to one of his eight cases, says, "It is questionable whether the case should be classified as spondylolisthesis". Radiographically it is not, yet in his summary he says, "the author reports eight cases".

Several base their diagnosis on the appearance of antero-posterior radiographs which, in my opinion, give no justification for such a diagnosis. Thus only one of the five sketches of the antero-posterior radiographs of Turner and Tehirkin shows the characteristic features illustrated by Jones and Lovett and Whitman. The other four sketches do not give any evidence of spondylolisthesis; further, they state that no lateral radiographs were made. This statement also appears in the same article: "As the condition generally develops in adults, the symptomatology is usually sufficiently striking to point to a correct diagnosis, although diagnostic errors have been made by Neugebauer and others." Herndon reported in 1927 that he had examined 941 patients suffering from back injuries, all but a few being coal miners, and found only two cases of spondylolisthesis.

After a careful examination of the radiographs of all the patients in my series of over three thousand, I have found only five definite cases of spondylolisthesis: three girls, ages 13, 15, and 17; a boy, age 17; and one man, age 35. In men I have found two cases in which the lumbosacral angle was much reduced—A. Whitman's 'pre-spondylolisthesis': one was due to a severe blow on the shoulder, the other to a fall of 16 feet (*see Figs. 515 and 516*). Yet many patients complained of low back pain, and had received injuries in mining and other heavy work; in fact, the series represents an examination of a large proportion of all the back injuries sustained in the factories and mines of a large industrial area, and several hundred soldiers who had had injuries

to the back from falls or crushes. These cases of spondylolisthesis came to my notice within a few days of each other, and but for the fact that I had been carefully looking for an example of this so-called common condition, I might have thought that I had missed many, and that, after all, it was common. Quite a number of the patients gave clinical signs suggestive of spondylolisthesis, but the antero-posterior and lateral radiographs failed to confirm the diagnosis.



FIGS. 493 and 494.

will be seen that the anterior border of the transverse process is continuous with the anterior border of the body. With a definite case of spondylolisthesis of the superior surface of the body of the 5th lumbar vertebra is facing forwards, and an antero-posterior radiograph of the patient will show the 5th lumbar body and transverse processes in this plane, and this characteristic outline (*Fig. 494*) of the anterior border of the body and transverse processes will be projected against the shadow of the sacrum. These features are well shown in the radiographs of the authors mentioned, and in *Figs. 495-498*.

When present such an appearance is indicative of spondylolisthesis. I have not seen any other condition which gives this appearance on an antero-posterior

X-ray Appearances.—It will be well at this stage to describe the X-ray appearances of the deformity.

Antero-posterior Radiographs.—These may be absolutely typical. Such radiographs are illustrated in the orthopaedic surgery text-books of Jones and Lovett (p. 342) and Whitman (p. 126). The antero-posterior radiographs of four of the cases referred to above, which are described later, show the same feature. The characteristic features of the radiographs of the 5th lumbar vertebra when X-rayed from above—that is, from its superior to its inferior surface—are shown by the radiograph of a disarticulated 5th lumbar vertebra (*Fig. 493*). It



FIG. 495.—Antero-posterior radiograph of Miss G. H., age 25, showing spondylolisthesis, and bone-grafts from the 3rd lumbar spine to the sacrum and ilium which were inserted in October, 1917.



FIG. 496.—Radiograph of Mr. C., age 35, showing spondylolisthesis, and a catheter in the ureter through which the pelvis had been filled with sodium bromide solution. Note hydro-nephrosis and stones.

radiograph. Antero-posterior radiographs of patients having a sharp lumbosacral angle or 'lipping' of the anterior margins of the vertebral bodies of the 5th lumbar and 1st sacral have been interpreted as spondylolisthesis. Antero-posterior radiographs illustrating these two conditions are shown in *Figs. 433* and *499*. The latter shows a dark crescentic line (indicated by an arrow)



FIG. 497.—Antero-posterior radiograph of Miss J. F., age 13, showing spondylolisthesis.



FIG. 498.—Antero-posterior radiograph of Miss P. S., age 15, showing spondylolisthesis.



FIG. 499.—Showing a condition which may be mistaken for spondylolisthesis.

which is due to ossification of the ligaments of the anterior surface of the 5th lumbar and 1st sacral vertebræ with some 'lipping' of the opposing borders. (Note also that the laminae of neither the 5th lumbar nor 1st sacral have fused, and a separate spinous process of the 1st sacral is shown.) The condition may be associated with massive new bone formation in front of the bodies, which may not only be misinterpreted owing to its radiographic appearances, but also owing to its appearance at post-mortem.

Lateral Radiographs.—The lateral radiograph furnishes us with almost absolute proof of the presence of any anterior 'slipping' of the 5th lumbar body on the sacrum. A good lateral radiograph will show the outlines of both these vertebræ. consequently the alinement of the anterior and posterior borders of the bodies can be seen. If a line is drawn connecting the anterior surfaces of these vertebræ, it will be seen to form a curve, the extent of the convexity depending on the lumbosacral angle, but if the 5th lumbar body has slipped forward on the sacrum, a definite break will be produced in the regularity of the curve. Further, if a straight-edge is placed along the shadow of the anterior border of the 1st sacral body, it will project upwards and forwards well to the front of the shadow of the 5th lumbar body, whereas if there is spondylolisthesis, the straight-edge will cut into the body of the 5th lumbar.

Bowman suggests that the alinement should be judged from the posterior surface of the bodies, but, as Le Wald has shown, the superior border of the 1st sacral laminae may be mistaken for the superior border of the 1st sacral body, and an erroneous interpretation of spondylolisthesis is made. On the other hand, one may be misled by judging the alinement solely from the anterior border. In one case in my series, while the antero-posterior radiograph showed what I have described as the typical appearance of spondylolisthesis, the

lateral radiograph appeared to show that the anterior alinement was perfect. With a better radiograph the cause of this misinterpretation was apparent. The 5th lumbar vertebral body had slipped forward and its anterior border had been brought into alinement with the shadow of the lateral border of the sacrum, which was interpreted as the anterior border of the middle of the sacrum. In this case it was the antero-posterior radiograph which gave the clue to the diagnosis. The moral of this is that a diagnosis should only be made on good radiographs taken in the antero-posterior and lateral planes. Particularly is it necessary to correlate the appearances shown on the radiographs taken in the two planes in cases where only a slight degree of slipping has occurred.

Figs. 500-502 are lateral radiographs showing degrees of spondylolisthesis. It will be noted that the greatest degree occurs in a girl of 13. *Fig. 500* is a lateral radiograph of Miss G. H., age 25, showing dislocation of the lumbar vertebræ on the sacrum and a bone-graft extending from the 4th lumbar spine



FIG. 500.
Miss G. H., age 25.



FIG. 501.
Miss P. S., age 15.



FIG. 502.
Miss J. F., age 13.

to the sacrum. *Fig. 501* is a lateral radiograph of Miss P. S., age 15. It shows a slipping of the 5th lumbar body, the lower border of which forms an angle of 90° with the anterior surface of the 1st sacral body, while its posterior body appears to be lying against the upper part of the anterior border of the sacrum. The laminae and spinous process appear to be 'drawn out' so that the latter projects beyond the spinous processes of the sacrum and the other lumbar vertebræ. The 4th lumbar laminae also appear to be a little elongated. The original radiograph shows that the laminae and spinous process are sclerosed, owing apparently to the pressure to which they are subjected. *Fig. 502* is a lateral radiograph of Miss J. F., age 13. It shows that the 5th lumbar body has 'slipped' forward to such an extent that its inferior surface is opposed to the anterior surface of the 1st sacral, the superior extremity of which appears to articulate with the inferior surfaces of the laminae, inferior articular processes, and spinous process, which show the corresponding curvature. The spinous process of the 5th is seen on the original radiograph to project further backward than the spinous processes of the upper lumbar vertebra and the sacrum. These radiographs and the photographs of the specimen in the Royal College of Surgeons Museum (*Figs. 510, 511*) are at variance with the opinion

of Lovett, who states that "the essential point is not so much the subluxation of the body, as the antero-posterior elongation of the body. This always exists and is apparently the pathological condition peculiar to this situation and this condition."

Etiology.—The variety of causes ascribed by different observers for this condition is sufficient evidence in itself to prove that either the cause is unknown or that it is brought about in many different ways. It will be seen from this brief summary that lesions of every structure entering into the formation of the lumbosacral joint have been blamed for the occurrence. Many of the observers believe in the same fundamental cause but attach importance to a particular feature. Neugebauer formulated the theory that it is a static or traumatic deformity of the spine favoured by the imperfect ossification of the laminae of the 5th lumbar vertebra. He also advanced two other theories: (1) That it is due to arthritis of the lumbosacral joint; and (2) That it is a pressure deformity of the normal vertebra resulting from prolonged physiological strain. Magnuson states that "it is a result of improper bony contact at the articulations and of a loss of bone continuity in the neural arch of the 5th lumbar." Willis and Hey Groves support this. Strasser considers that it is due to a primary arthritis of the lumbosacral articulations. Chian says that abnormal relations of the lumbosacral inter-articular processes due to defective ossification bring it about.

Barnes favours a solution of continuity across the neural arch of the 5th lumbar between its superior and inferior processes on either side. Herrgott thinks it due to inflammatory change following a breach of the vertebral arch by trauma. Braun, Swartz, and Speth all look upon congenital anomalies as the cause. Rokitsanski suggests atrophy of the articular cartilage. Lane and Roberts consider it due to excessive weight-bearing which produces thinning and elongation of the neural arch.

Kleinberg and Fitch consider that the condition is due to trauma. Kleinberg is of the opinion that there is an initial developmental defect in the lumbosacral region affecting the body, and more particularly the ligamentous structure, in these cases; also that the position of the lumbar articular processes in the sagittal plane favours a dislocation; but he believes that trauma is the chief factor in the etiology. Darling says it is due to trauma favoured by congenital defect; that it has a gradual onset; and that the symptoms at the time of the injury may be few. Palmer blames the tearing of the ligaments. Lamb says it is probably due to a congenital malformation remotely connected with foetal hydrorrhachis, traces of which remain in the form of widening of the medullary ring. Albee holds trauma responsible, and states that the body of the vertebra is chiefly affected, whereas the laminae and spinous processes may remain practically in place. It may be subluxated in part, or its body and anterior half of its arch may be dislocated while the spine and inferior articular surface and the posterior half of the arch may remain in place, in time even be finally ankylosed to the sacrum. Bowman and Goin say it is usually the result of trauma—the injury often being considered of minor importance. They state that "we believe spondylolisthesis is a much more common lesion than it is generally thought to be; that many of them reduce themselves spontaneously, and that many cases of so-called

sacro-iliac slips are in reality cases of unrecognized spondylolisthesis." Most of their patients were men, but the two cases showing the worst deformity were women. The following two cases cited by them are interesting:—

1. A woman, age 65, who awoke one morning with severe pain in her back radiating down the left thigh posteriorly. A herpes developed as the pain continued; three grains of morphia were required daily to relieve the pain. Neurological diagnosis was "spinal cord tumour at the level of the 4th lumbar". X-ray examination revealed a spondylolisthesis of the 4th lumbar vertebra. The symptoms were entirely relieved by correcting the spondylolisthesis.

In support of the statement which they make—that in this condition it is quite easy to replace the vertebræ with a moderate amount of manipulation, and that many patients find relief in the hands of chiropractors, osteopaths, and other manipulators—they quote the following case:—

2. A man, age 36, while lifting a section of pipe had a sudden severe pain in the lumbosacral region and was unable to straighten. X-ray examination of the spine revealed a spondylolisthesis of the 5th lumbar. The pain was severe and he was taken to hospital, where it was decided to attempt reduction under anæsthesia. While the surgeons were gently manipulating his legs in an attempt to work out the necessary motions prior to the induction of the anæsthesia, the patient suddenly cried out, and after a few tentative twistings announced that he was cured and the anæsthesia and surgical procedure could therefore be dispensed with—in fact he declined any further attention and went home.

Eden says "the change which occurs is not a dislocation but a displacement due to lengthening and bending of the inter-articular portion of the last lumbar vertebra, as a result of which the vertebral column becomes displaced forwards and downwards. The 1st sacral vertebra becomes gradually torn away by pressure and ankylosis eventually occurs in the morbid position." Nathaniel Mills considers that the asymmetry of the articular processes of the lumbosacral joint renders the joint unstable, so that sudden rotation may cause fracture or dislocation in this area. Hibbs confirms this opinion, and states that a rotation deformity more marked on one side than the other is always found.

Henry, who states that he had had five cases in adult males in a year, believes that it is due to the trauma of modern industry and for that reason is more common in men than women. He is of the opinion that trauma presumably shears off the superior processes of the sacrum. All his cases showed nerve involvement. He says the lesion is a progressive one. First, a slight forward luxation of the 5th lumbar vertebra; gradually the ligaments stretch and the dislocation progresses, increasing the instability. He says that three of his cases showed congenital anomalies of the articular processes, which face directly upwards. Rose and Carless state that "It arises from fracture of the articular processes of the lumbosacral synchondrosis, or from imperfect development of the laminae or pedicles of the lowest lumbar vertebra, as a result of which the pressure of loads carried on the shoulders or the weight of a pregnant uterus brings about the displacement."

Wollenberg illustrates a congenital forward dislocation of the vertebræ on the 2nd lumbar, which is smaller than the other bodies. He states that the mother and sister of the patient showed a similar defect. Von Lackum says

that the thinness of the posterior part of the intervertebral disc diminishes its effectiveness as a shock absorber, while the complete absence of spinous processes from the sacrum, with thinner and longer ligaments, increase instability. Ayers considers that spondylolisthesis probably results from the wearing away of the articular facets in a patient suffering from an exaggerated lumbosacral angle.

CASE REPORTS.

The short descriptions of the following five cases and one specimen show that spondylolisthesis is due to several causes.

Case 1.—Miss P. S., age 15.

PHYSICAL SIGNS.—*Figs. 503-505* show the deformity due to spondylolisthesis. The most obvious feature shown in *Fig. 503* is the prominence of the sacrum (which appears to be pushed into a more superficial position), and a prominent knuckle at the upper part of this projection. A careful study of the lateral radiograph



FIG. 503.—Case 1.



FIG. 504.—Case 1.

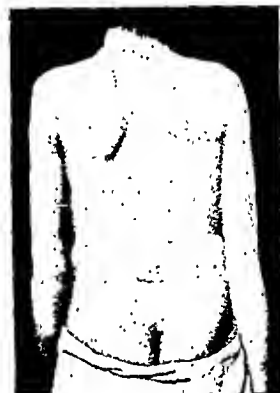


FIG. 505.—Case 1.

(*Fig. 501*) shows that this upper and most prominent spine is the spine of the 5th lumbar vertebra which has been compressed between the superior surface of the sacrum and the 4th lumbar spine. Immediately above this prominent knuckle the spines are not so prominent, but they can be seen and felt. In the dorsilumbar area the spines appear a little more prominent than in the upper part of the column. *Fig. 504* shows the extent of flexion of the spine permitted by the deformity. The radiograph of the posterior surface (*Fig. 498*) shows a suggestion of a transverse furrow running forward over the iliac crest and an obliteration of the median furrow which normally ends at the 3rd sacral spine and of the surface indications of the posterior superior spines of the ilium.

SYMPTOMS AND HISTORY.—The patient had never experienced any pain or discomfort from the dislocation. No evidence could be obtained from her or her mother that she had ever received injury. Attention was drawn to the deformity by the family doctor during an examination necessitated by an attack of infectious disease six months previously.

RADIOGRAPHS.—The antero-posterior and lateral radiographs (*Figs. 498* and *501*) show the characteristic features of spondylolisthesis. It will be seen that the displacement is symmetrical and that no scoliosis has been produced. The neural arch and spinous processes do not show any evidence of non-fusion with the body. The appearance suggests a similar condition to the specimen illustrated in *Figs. 510* and *511*.

Case 2.—Miss G. H., age 25.

PHYSICAL SIGNS.—*Figs. 506 and 507* show the deformity present ten years after bone-grafts had been inserted. No photographs are available which would illustrate the condition before operation. The scar of the incision is shown and the prominence of the 'superficial' sacrum. No definite transverse furrows are shown, but

there are several 'dimples' in the upper lumbar region.

SYMPTOMS AND HISTORY.

—The patient says that she never had any pain, but she 'waddled'. Before the operation she could turn her right leg so far round that the toes faced backwards. She gave a history of a fall on the stairs when she was young.

RADIOGRAPHS.—The antero-posterior and lateral radiographs (*Figs. 495 and 500*) show the characteristic features of spondylolisthesis. The 'slipping' has not been quite symmetrical and there is a slight scoliosis. Two bone-grafts are shown—one from the 3rd lumbar spine to the

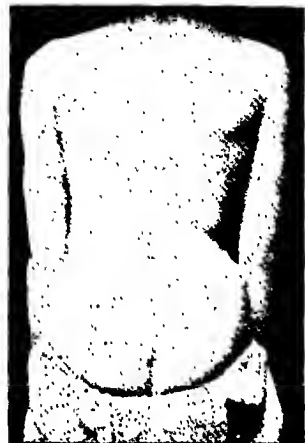


FIG. 506.—Case 2.



FIG. 507.—Case 2.

3rd sacral spine, and one from the 3rd lumbar spine to the ilium at the level of the sacro-iliac joint. The lower end of the latter graft has expanded but not united with the ilium and has formed a false joint. Radiographs taken before the operation do not suggest any solution of continuity of the neural arch, which appears to be well developed.

Case 3.—Miss J. F., age 13.

PHYSICAL SIGNS.—I have been unable to take photographs illustrating her deformity. She is of stout build, and marked transverse furrows suggest a shortening of the lumbar spine. The back shows a scoliosis with the lumbar curve to the left. The sacrum is prominent; the most prominent spine, as shown in the lateral radiograph (*Fig. 502*), is that of the 5th lumbar vertebra. Immediately above this prominent spine the median furrow begins in a marked depression. On flexion, which is limited, there is a general prominence of the dorsal spine on the right side. The other movements of the spine are normal.

SYMPTOMS AND HISTORY.—No pain or history of any injury. The mother says that the patient did not walk in a normal manner when a child; she always had difficulty in bending forwards with her knees straight.

RADIOGRAPHS.—Antero-posterior and lateral radiographs (*Figs. 497 and 502*) show the characteristic features of spondylolisthesis. The 'slipping' appears from the antero-posterior radiograph to have been symmetrical, but there is a scoliosis of the lumbar spine. They show no evidence of solution of continuity of the neural arch, which appears to be well developed.

Case 4.—Mr. C., age 35.

PHYSICAL SIGNS.—*Figs. 508 and 509* show that the patient is of short, stout stature. The photograph of his back shows that the median furrow ends at the lower lumbar region, but the sacrum does not appear very prominent. There is no sign of any knuckle kyphos. Transverse furrows above the iliac crests indicate some shortening of the lumbar spine. *Fig. 509* shows that flexion of the spine is not much impaired; he can touch his toes without bending his knees. It also shows a very slight dip in the bend at the level of the lumbosacral joint.

SYMPTOMS AND HISTORY.—The patient could not give any history of injury

to the spine and had never felt any pain which appeared to be connected with it. At the age of 17 he took up 'physical culture', which he later practised as a professional. Two years ago, when he had symptoms of stone in the kidney for which he was X-rayed, a collection of stones was shown in the right kidney. A

pyelograph was made (*Fig. 496*) which demonstrates a hydronephrosis and a collection of stones in the pelvis and calices of the kidney, also an opaque catheter in the ureter which shows the relation of the ureter to the displaced 5th lumbar vertebra.



FIG. 508.—Case 4.

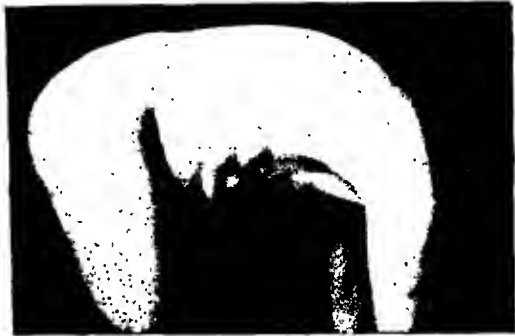


FIG. 509.—Case 4.

RADIOGRAPHS.—The diagnosis of spondylolisthesis was not made until I was recently examining the radiographs, which showed the characteristic shadow of the 5th lumbar vertebra. A lateral radiograph was taken to confirm this diagnosis, and this shows a complete dislocation of the 5th lumbar spine on the sacrum. The radiograph does not indicate any evidence of solution of continuity of the neural arch, which appears to be well developed. It had never been suggested to the patient before—even though he was often seen stripped for his gymnastic performances—that he had any deformity of the spine.

Case 5.—Mr. J. S., age 17.

PHYSICAL SIGNS.—I have been unable to obtain photographs or further radiographs of this patient, as he has left the district. He is of slender build and shows slight transverse furrows with a scoliosis of the lumbar spine to the left and a compensating curve in the dorsal region. He is tender over the lumbar and sacral spine and in the right iliac fossa. He walks with a limp and cannot sit up without external rotation of the right hip. The sacrum is prominent, and the lateral radiograph shows that the upper extremity of the prominence is due to a projection of the sacrum and that there is no spinous process of the 5th lumbar.

HISTORY AND SYMPTOMS.—He complains of pain in his right hip-joint. Two years ago he says he fell off his cycle and injured the right hip, which caused him to remain in hospital for ten months.

RADIOGRAPHS.—The antero-posterior radiograph is not very good, but it shows a faint outline of the characteristic border of the 5th lumbar body with poor development of the neural arch and failure of fusion of the laminae. The lateral radiograph shows a complete dislocation of the 5th lumbar on the sacrum.

Case 6.—Specimen of the lumbosacral region of a spine in the Museum of the Royal College of Surgeons.

Figs. 510 and 511 illustrate the two sides of the specimen. The cut surface shows a forward dislocation of the lumbar spine on the sacrum and a heaping up of the lumbosacral intervertebral disc behind the body of the 5th lumbar. The neural canal widens gradually from the level of the 2nd lumbar vertebra

to the lumbosacral joint, and with this widening the laminae are drawn out into long processes which in the photograph appear darker than the other bone, owing to an artificial red staining of the processes for museum purposes.



FIG. 510.—Case 6.

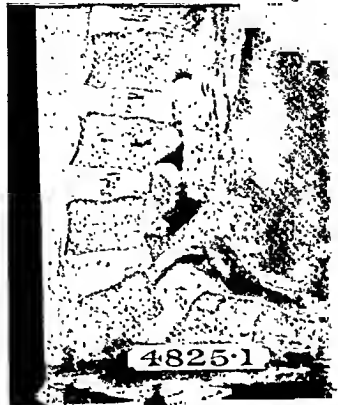


FIG. 511.—Case 6.

The spine of the 5th lumbar is shown to project posteriorly further than the spinous processes of the vertebrae above or below, and there is no suggestion of poor development of the neural arch.

HISTORY.—The history states that there was no evidence of any injury. This widening of the neural canal

and the 'drawing out' of the laminae suggest that if the condition is due to trauma, the injury must have taken place in early life.

A review of the features of these cases shows that the lesion occurs early in life, apparently without any symptoms, and may never be diagnosed unless for some reason it is suspected. To the obstetrician this is of great importance, for the deformity will prevent normal delivery. Its presence will be suggested to him by an examination of the 'rhomboid or lozenge of Michaelis'. This is indicated on the surface by the oblique furrows formed by the medial borders of the glutei maximi, which come together in the mid-line distally and appear to end in the shallow depressions over the posterior superior spines of the ilium on either side. The upper borders of the rhomboid are formed by lines joining the latter depressions to the depression usually present over the 5th lumbar spine. The essential measurements of the rhomboid are the distances between the posterior superior spines of the ilia and the vertical distance from the mid-point of the line joining these two points to the 5th lumbar spine. The average of the former in the normal is 4 in., of the latter $1\frac{1}{2}$ to $1\frac{3}{4}$ in. It will be seen from the photographs that the median depression which marks the 5th lumbar in the normal is not present in those patients having the deformity.

In only one case is there a definite faulty development of the neural arch, and in this there is a history of a severe injury. This and the other features described suggest that trauma is the probable cause and that the injury is often sustained in early life—even at birth.

Spondylolisthesis and other conditions which narrow the pelvis lead to great difficulty in obstetrics, and the child is sometimes so forcibly pulled, crushed, twisted, or turned that the skull, ribs, or limbs are broken or birth palsies are produced. Therefore, while spondylolisthesis in the mother leads to difficulty in childbirth, difficulty in childbirth may lead to spondylolisthesis in the child.

Physical Signs and Symptoms.—Two types of spondylolisthesis have been

described: (1) Unilateral, which is said to be accompanied by pronounced early paralysis due to compression and displacement of the corda equina. (2) Bilateral, which is a gradual process only accompanied by mild symptoms—the common type.

Pain.—Pain may be absent altogether. This was so in four of the cases I have reported. Nerve disturbances, which may be temporary, permanent, or recurrent, have been described by Ryerson and others. One of the patients mentioned above had a hydronephrosis with stones, but it was impossible to say what relation this bore to the dislocation, as the latter was not suspected at the time of the operation. Creyssel describes a case of spondylolisthesis in which adhesions around the dislocation involved the ureter and produced a hydronephrosis.

Limitation of Movement.—Flexion of the spine is usually restricted; this was so in four of my cases, but in one there appeared to be no restriction.

Gait.—Two of the five patients showed no abnormality, two had a slight waddle, and one a marked limp. Lovett describes the gait as resembling that of a quadruped.

Posture.—Trunk shortened. This may be shown by transverse furrows which encircle the body between the ribs and the iliac crests, diminution of the space between the ribs and the iliac crests, and between the ensiform cartilage and the pubis.

The sacrum is prominent, and, as shown in four of my cases, there is a still greater prominence of the 5th lumbar spine, with a depression above. In the female a vaginal examination will show a diminution of the true conjugate or antero-posterior diameter of the pelvic inlet. Only one patient had any difficulty in extending the legs—this is suggested as one of the signs of the displacement. Some authorities state that the abdomen is pendulous and the buttocks are prominent; this was not seen in any of my cases.

Differential Diagnosis.—Two of the five patients were diagnosed as tuberculous caries of the spine owing to the localized kyphos deformity. Radiographs and photographs of patients showing the deformity produced by tuberculous caries of the 5th lumbar and 1st sacral vertebrae are shown in Figs. 542-544 and 548. As a rule, with tubercle, pain is a prominent early symptom, often before any sign of a deformity, whereas in these cases of spondylolisthesis four had no pain. The prominence of the sacrum is not observed in tubercle. Radiographs will give conclusive evidence of spondylolisthesis or tubercle in the cases with deformity. Congenital deformities of the lumbosacral spine may also produce kyphosis; the nature of these will be shown by the radiograph.

Treatment.—Ryerson first reported a case of spondylolisthesis treated with a bone-graft. Albee immobilized the spine by bone-graft in eight cases. He says "the 4th and 5th lumbar vertebrae tend to pull themselves away from the graft and in an antero-posterior direction: the graft being inserted into their spinous processes is firmly pressed against the posterior surface of the sacrum." Cases have also been treated by Hibbs's method of lumbosacral fusion. Bowman and Goin consider that many cases of spondylolisthesis are reduced spontaneously. The authorities who either do not favour the open operation or who cannot obtain permission to operate, treat the patient by

spinal supports, exercises, and massage. Turner and Tehirkin state that "It is doubtful whether operative measures can aid conservative treatment in alleviating the symptoms and arresting the progress of the ailment." A study of the specimens and radiographs in my cases negatives the suggested possibility of manipulative reduction of the displacement.

V. DEFORMITIES DUE TO INJURY.

Deformities of the lumbosacral region of the spine due to fracture are not very common. When such a fracture does occur it is owing to very severe trauma. In the previous pages of this paper it has been indicated that trauma may produce a narrowing of the lumbosacral angle and spondylolisthesis, but the commonest fracture of the spine is the compression fracture of the vertebral body. This type is chiefly seen in the 5th and 6th cervical and the 12th dorsal and 1st lumbar. The fracture is usually due to forced hyperflexion, but the force necessary to produce it appears to vary greatly in different individuals. It is seldom that this type of fracture is seen in the lower lumbar region. The clinical and radiographic evidence will vary with the degree of trauma and the condition of the bone. As a general rule it may be said that the more obvious the clinical deformity, the greater the bone or joint abnormality shown on the radiograph.

In some cases if the patient is examined in the first few days, little sign of the deformity may be seen. The patient will usually complain of pain and tenderness in the region of the fracture and there may be obliteration or departure from the normal antero-posterior curvature. Antero-posterior and lateral radiographs may not show any definite bone deformity. Gradually with the absorption, organization, and compression of the damaged tissues,

the deformity begins to show. Several patients were radiographed within a few hours of their injuries and the pictures show little sign of compression of the vertebrae, but a week or so later a definite wedge-shaped compression of the vertebral body could be detected on the lateral radiograph (*Fig. 512*); no attempt had been made in the interval to prevent movement. This rather suggests that a greater degree of deformity will result if the injured vertebral body is not relieved of all compression during the process of repair. Such fractures may be produced in old people with relatively trifling injury, and therefore may be quite unsuspected until perhaps a definite kyphosis is seen. If this has taken several weeks to develop, its appearance may not be related to the injury and may give rise to a suspicion of pathological destruction of the bone—tubercle or neoplasm, for



FIG. 512.—Lateral radiograph showing wedge-shaped compression fracture of the body of the 12th dorsal vertebra due to sudden hyperflexion of the trunk.

example. It is probably owing to this delay in the production of the deformity that this appearance of the vertebral body has been described as Kummel's disease, which is defined as a non-tuberculous rarefying osteitis of the vertebral bodies accompanied by softening and collapse of the bodies.

In some patients who have had injuries to the back, the bone damage is not so extensive and deformity may not be seen for several months. This is due to the fact that the injury has chiefly involved the ligaments and the intervertebral discs; and the bones preserve their normal positions. Gradually the damaged intervertebral discs contract, sometimes more on one side than the other, the vertebral bodies are brought into closer contact with one another, and as they are not so well protected by the buffer action of the disc, they are subjected to more strain. This causes a sclerosis and 'lipping' of the margins of the vertebral bodies and often a calcification of the connecting ligaments, which can be shown on radiographs of such injuries. If the damage has been greater on one side, a curvature will develop, which often gives rise to a compensatory curvature in the vertebræ above. The opposing borders of the vertebral bodies on the concave side of these curves will undergo a similar process. *Fig. 513* shows the bone and joint changes five years after an accident which damaged the lumbosacral joint. It shows a sclerosis of the bone in the neighbourhood of the left side of the lumbosacral joint and ossification of the connecting ligaments. There is also sclerosis and lipping of the bodies of the 2nd and 3rd lumbar vertebræ on the right side—the concave side of the curvature.



FIG. 513.

Fig. 514 is a radiograph of a man, age 71, who gave a history of an injury to the lower part of his back thirty years before. He is now complaining of 'lumbago' and 'sciatica'. This radiograph shows similar features to *Fig. 513*, but the 'lipping' is more marked. Jones and Lovett have pointed out that "in all cases of spinal fracture, however slight, there is a history of trauma, whether direct or indirect; but it must be remembered that the effects are not necessarily immediate and may reach their maximum a good while after the injury."



FIG. 514.

The laminae and spinous processes of the lumbar vertebræ are stronger and better protected against injury than the cervical and dorsal vertebræ. consequently fractures of these processes are rare unless they are part of a more extensive injury. They have been reported in cases of traumatic spondylolisthesis and as a result of direct blows over these processes. Such fractures in some cases cause compression and damage to the

corda equina and paralysis may follow. *Fig. 516* shows a fracture of the laminae of the 5th lumbar vertebra in a man, age 36, who fell 16 feet from a tree and landed on his buttocks. Radiographs showing non-fusion of the laminae might be interpreted as fracture of the laminae (see *Figs. 457 and 458*).

Balensweig cites two cases :—

1. A labourer, age 45, slipped down some steps whilst he was carrying a load of wood. Subsequent radiographs showed a forward dislocation of the 4th lumbar on the 5th.

2. A carpenter, age 54, fell 14 feet and landed on his buttocks. Twelve months after he complained of pain in the lower lumbar region which radiated down the left leg. The movements of his spine were restricted, especially extension; and there was a loss of the normal lumbar curvature. No angulation or depression could be seen above the sacrum. He was tender in the gluteal region and along the course of the sciatic nerve. A lateral radiograph showed a displacement of the lumbar spine on the sacrum of about $\frac{3}{16}$ in. A bone-graft was inserted: the patient has since developed a radiculitis probably due to osteo-arthritis, and is now incapacitated.

Figs. 515 and 516 are antero-posterior and lateral radiographs showing 'pre-spondylolisthesis'. *Fig. 515* shows sclerosis of the pedicles and the right superior and inferior articular processes of the 5th lumbar vertebra



FIG. 515.



FIG. 516.

with little upward displacement of the latter and approximation of the spinous processes. *Fig. 516* shows a sharp lumbosacral angle and gives the suggestion of a slight dislocation of the lumbar vertebrae on the sacrum. The laminae show an irregular fracture line which indicates a separation of the superior from the inferior articular processes,

all of which exhibit some irregularity. The spinous process of the 5th lumbar vertebra is on a higher plane relative to the vertebral body than is the case in the normal. This appearance is quite different from the abnormality demonstrated in *Fig. 443*. This patient, a man, age 35, fell 16 feet from a tree and landed on his buttocks ten years ago. He now presents a slight lumbar curvature and complains of pain in the lumbosacral region.

Fracture of the transverse processes occurs from direct and indirect injury; they are frequently fractured by hyperflexion of the trunk. Sudden unopposed contraction of the quadratus lumborum is said to produce a fracture of these processes—certainly fractures become more obvious radiographically by the pulling apart of the broken extremities with contraction of the quadratus lumborum. The 3rd lumbar transverse processes are most frequently fractured; this may be due to their greater length. The costal element of the transverse processes of the 1st lumbar vertebra is sometimes formed from a separate epiphysis which fails to fuse with the body of the vertebra and forms a lumbar rib. This has been interpreted from the radiograph as a fracture of the transverse process. I have pointed out that the costal element

of the 5th lumbar vertebra which is developing sacral characters has also a separate centre of ossification, and a line indicating that this has no bone union with the vertebral body may be shown on the radiograph and lead to an erroneous interpretation of fracture. The shadow of the lateral border of the psoas as it passes over the transverse processes, and the difference in density of the main mass of the process and its thickened lateral extremity, may also give a false impression of fracture. *Fig. 517* illustrates a fracture of the iliac crest and the transverse process of the 4th lumbar vertebra with an irregular process of new bone attached to the 4th transverse process and articulating with the iliac crest. This new bone formation followed a septic infection of the wound which occurred seven years before this radiograph was taken.



FIG. 517.



FIG. 518.

a week. She had some paresis of the lower extremities and incontinence, but the condition has improved and she is still alive and well enough to be wheeled about after two years.

Figs. 519-521 are pictures of a miner, age 40, who was crushed by a heavy mass of coal which fell

With a complete fracture-dislocation of the spine, the upper part of the vertebral column is usually displaced forwards on the lower, and the spinal cord or *corda equina* is stretched over the top of the body of the vertebra heading the lower column, and between it and the arch of the vertebra which was above. *Figs. 518-520* are examples of the serious types of fracture which are met with in this area. *Fig. 518* shows a fracture-dislocation through the 1st lumbar vertebra with a lateral displacement of the fragments. This patient was thrown out of the side-car of a motor cycle by a collision with a car. She sustained in addition a fracture of one femur and of the base of the skull. The spinal deformity was not well marked, and owing to this and her other injuries it was not discovered for over



FIG. 519.



FIG. 520.

on his shoulders as he was working in the mine. The radiographs show that there was a complete separation of the innominate bones from one another and a vertical fracture of the right side of the sacrum, with an upward displacement of the right side; also fractures of the left transverse processes of the 2nd, 3rd, and 4th lumbar vertebræ. *Fig. 521* shows his present condition and deformity; the buttock is seen to be much higher on the right side than the left. He had no bladder symptoms and is able to do regular light work.



FIG. 521.

Bowman reports that in his experience compression fractures of the vertebral bodies occur in the following order: 1L, 12D, 2L, 11D, 5L, 5 and 6C. Herndon found fractures of the vertebræ in 71 out of 941 cases of injuries to the back; 41 involved the vertebral processes; 30 involved the vertebral body, and 12 of these were 1st lumbar vertebræ. Von Lackum states that in a review of the fracture-dislocations of the spine at the New York Orthopedic Dispensary and Hospital during the last twelve years it was found that the majority of those lesions took place in the lumbosacral region.



FIG. 522.

Fig. 522 is a radiograph showing 'lipping' of the left borders of the 2nd and 3rd lumbar vertebræ. The patient gave a history of an injury to this part of the back two years before.

VI. DEFORMITIES DUE TO PATHOLOGICAL CHANGES IN THE BONES AND JOINTS.

The pathological changes which produce deformity are caused by: (1) Organisms of acute infections such as staphylococci, streptococci, pneumococci, gonococci, and typhoid bacilli. (2) Organisms of chronic infections—tubercle, syphilis, actinomycosis. (3) Neoplasms—simple and malignant. (4) Deficiency diseases and other bone diseases such as rickets, Paget's disease, and osteomalacia.

ACUTE INFECTIONS.

Osteomyelitis.—Acute osteomyelitis of the spine is fatal in the majority of cases; in the survivors the degree of deformity will depend on the amount of bone and ligament destruction. As a rule there is no great loss of tissue bulk, and as the condition enforces almost absolute rest of the spine, deformities due to the weight of the trunk on the damaged bone do not arise. The chief deformity seen on a clinical examination is rigidity of the part of the spine involved, owing to bone ankylosis of the intervertebral joints, and ossification of the ligaments. Greater deformity results if any

strain is placed upon the parts before healing is complete. Radiographs will not show any evidence of acute osteomyelitis in the early days; later, rarefaction of the bones may be seen; later still, erosion of the bone. Those cases in which a chronic inflammatory process persists will show a varying amount of bone destruction and, if healing is taking place, sclerosis of the surrounding bone. Sequestra may be shown and can be recognized because the dead bone retains its calcium and therefore its opacity to X rays, whereas the living bone is partly decalcified owing to the presence of toxins and the disuse necessitated by the lesion. Further, if the condition has originated in a fracture and some of the detached fragments of bone have died, they will retain their sharp fracture contour; the borders of the living bone will be round.

Fig. 523 represents the lumbosacral area of a patient, age 45, who had a gunshot wound of the right side of the lumbosacral joint ten years ago. A sinus persisted which healed over from time to time. One year ago an abscess was opened in this area. The picture shows some irregularity of the right side of the lumbosacral joint and sclerosis of the surrounding bone. An irregular, curved, slender sequestrum can also be seen which appears to lie parallel to the crest of the ilium. The anterior common ligament is ossified and the left transverse processes are irregular and pointed owing to ossification of the iliolumbar ligament and fascia attachment.



FIG. 523.

Whitman gives the following tables showing the situation of the lesion in acute osteomyelitis of the spine in 61 collected cases:—

Cervical region	12
Thoracic	15
Lumbar	24
Sacral	10

He states that in 30 of 56 cases reported by Grisel the patient died of general infection, pleuropneumonia, or meningitis before the diagnosis had been made and before an abscess developed.

			Recovered	Died
Suboccipital	1	4
Cervical	2	2
Dorsal	7	3
Lumbar	13	15
Sacral	0	6
			23	30

Arthritis.—As a complication of several of the infectious diseases—small-pox, typhoid, yaws, dysentery, pneumonia, scarlet fever—arthritic conditions may arise. Most of these completely resolve, but when associated with pyogenic infection of the joint, necrosis of bone is liable to occur, or

destruction of the articular cartilage with resulting bone ankylosis and spinal curvature.

Occasionally, as a complication of gonorrhœa, practically all the articular surfaces of the spine fuse together to form a fixed rigid column, as if some osteoplastic substances had been secreted into the joint which had welded the surfaces together without any evidence of bone destruction or overproduction from the external surfaces. Such patients present a general rigid kyphosis. *Fig. 524* shows fusion of all the articulations of the lumbar and sacral joints. The original radiograph shows a general uniform rarefaction of



FIG. 524.

all the bone, which appears to have lost its normal striae. There is no evidence of ossification of the anterior common ligament. The patient gave a history of acute rheumatism following an attack of gonorrhœa two years previously. Verrall says that gonorrhœa is responsible for 65 per cent of all cases of severe arthritis with rigid spine, and that about 30 per cent are due to absorption of toxic products from the alimentary tract.

Typhoid spine is rare. The complication is most common in young adult males in an estimated proportion of 1 to 1800 cases of typhoid fever. In 36 per cent of 53 cases of typhoid spine investigated by Silver, local deformity indicated a destructive process (Whitman).

With complete bone ankylosis of the spine, the lumbar spine is flat, the dorsal spine forms a marked regular kyphosis, the curve of which is continued into the cervical area. Such a patient has no rotation of the spine and consequently has to turn his whole body if he wishes to look behind him, as in the type described by Bechterew. In those cases in which in addition ankylosis of the hips and shoulder-joints occurs the patient will have difficulty in retaining his balance when standing without supports. This type is described as the Marie-Strümpell variety.

A similar appearance to that seen in *Fig. 524* is often shown on the radiographs of patients who have suffered from a non-specific arthritis. The cause of this type of arthritis is generally thought to be an absorption of toxic products from some septic focus—sinuses, teeth, alimentary tract, gall-bladder, for example. Other authorities suggest that it might be a deficiency disease. *Figs. 525* and *526* are antero-posterior and lateral



FIG. 525.



FIG. 526.

Figs. 525 and *526* are antero-posterior and lateral radiographs of the lumbosacral area of a man, age 49, nine years after an acute arthritis. They show ossification of the anterior ligament, interspinous

